The Day-to-Day Associations Between
Sleep Characteristics, Affect, and Affect Reactivity

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Studies on healthy adults reveal either unclear or inconsistent results regarding the proximal, day-to-day relationship between sleep characteristics (sleep duration, continuity, timing) and different dimensions of mood (positive affect, PA, and negative affect, NA). In addition, while experimental evidence suggests that sleep changes can impact mood by exaggerating people’s emotional response to environmental factors, few studies have tested whether these findings generalize outside the laboratory. The current study aimed to examine 1) a bidirectional model of sleep and mood, and 2) the effects of sleep characteristics on affect reactivity, a measure of emotional response to daily experiences. Participants were healthy, midlife adults (30-54 yrs old, N =462) drawn from the Adult Health and Behavior Project- Phase 2 study. Across a 4-day monitoring period, sleep characteristics were measured via actigraphy and ecological momentary assessment methods were used to collect repeated measures of participants’ affect, work and social experiences. Affect reactivity was quantified as momentary changes in affect following these experiences. Using hierarchical linear modeling, we tested whether participants’ sleep characteristics on a given night predicted next-day affect and vice versa, and we tested whether sleep characteristics influenced affect reactivity. We found higher levels of PA predicted later sleep timing (B =.23, p =.012), but there were no other significant associations between sleep characteristics, PA and NA (p’s >.05). Sleep characteristics did not moderate the effects of daily experiences on either PA or NA (p’s >.05). There were significant individual differences in several
of the relationships between sleep, affect, and affect reactivity (p’s < .05). Overall, our findings suggest that day-to-day fluctuations in behavioral sleep patterns generally do not associate with subsequent affective experience. There may be graded and cumulative effects of sleep disruptions on affect and affect reactivity that are not observed in the context of small, daily fluctuations in sleep characteristics.
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1.0 Introduction

Sleep is vital for the processing and regulation of emotions, and the relationship between sleep and mood has long been documented, with evidence associating various forms of sleep disturbances with increased risk for depression (Baglioni, Spiegelhalder, Lombardo, & Riemann, 2010; Deliens, Gilson, & Peigneux, 2014; Jansson-Fröjmark & Lindblom, 2008; Johnson, Roth, & Breslau, 2006; Ohayon & Roth, 2003; Sivertsen et al., 2012; Taylor, Lichstein, & Durrence, 2003; Walker & van Der Helm, 2009). Consistent with this evidence, studies show that treatment of sleep difficulties can simultaneously reduce depressive symptoms (Manber et al., 2011; Manber et al., 2008; Taylor, Lichstein, Weinstock, Sanford, & Temple, 2007). These findings suggest a link between sleep and mood, but what remains unclear is how specific components of sleep relate to mood and the directionality of these associations. Identifying the proximal, temporal nature of these associations, that is the day-to-day covariation between sleep and mood, and their underlying mechanisms can potentially shed light on the development of co-occurring sleep and mood problems, and thereby inform targeted interventions.

The relationship between sleep and mood may be bidirectional, such that within-person changes in sleep lead to changes in mood, and changes in mood likewise predict changes in sleep. Experimental work shows that among healthy adults, changes in various behavioral characteristics of sleep alter mood. Specifically, after people experience restricted sleep duration, shifted sleep timing (earlier or later), or poor sleep continuity (e.g., frequent awakenings), they tend to report more negative mood and less positive mood the following day (Dinges et al., 1997; Kahn, Fridenson, Lerer, Bar-Haim, & Sadeh, 2014; Taub & Berger, 1976). Changes in sleep characteristics may impact mood by modifying how people emotionally respond to environmental
factors (Franzen, Siegle, & Buysse, 2008; Gujar, Yoo, Hu, & Walker, 2011; Tempesta, Couyoumdjian, Curcio, Moroni, & Marzano, 2010). On the other hand, physiological and cognitive arousal, both of which can stem from anxious and depressed mood, can subsequently alter an individual’s nighttime sleep characteristics (Tang & Harvey, 2004; Zoccola, Dickerson, & Lam, 2009). As further described below, we posit a bidirectional model whereby changes in sleep characteristics alter a person’s subsequent mood, in part by impacting their ability to regulate their emotional responses to daily experiences, and changes in mood perpetuate changes in his/her sleep patterns (Figure 1). With the onset of sleep or mood problems, such a feedback loop may underlie the development and maintenance of syndromal sleep and mood symptoms.

One approach to test the temporal relationship between sleep and mood is to test whether sleep on a given night predicts mood the following day, and whether mood on a given day predicts sleep that night. Because persons with sleep or mood disorders often already have co-occurring symptoms and take medication, focusing on healthy adults allows researchers to study the temporal relationship between sleep and mood patterns unconfounded by these factors. Studies using this design have begun to show a possible bidirectional relationship between sleep and mood. To provide scaffolding for a review of this literature, the following sections will first define sleep characteristics, mood and affect, describe factors that regulate each of these processes, and outline a proposed bidirectional model.
1.1 Mood, Affect, and Sleep

1.1.1 Sleep Characteristics

Sleep is a complex process and one approach to understand the sleep-mood relationship is to identify how specific dimensions of sleep influence mood states. Sleep can be characterized on a physiological, behavioral, and subjective basis. Several behavioral dimensions of sleep include sleep duration, sleep timing, and sleep continuity (Buysse, 2014). Sleep duration refers to the total length of time (i.e., hours) that an individual sleeps in a single period. Sleep continuity refers to disruptions (or lack thereof) to an individual’s sleep period and takes into account awakenings and time it takes to fall asleep (i.e., sleep latency). Lastly, sleep timing refers to the time period of an individual’s sleep within the 24-hour day. Among healthy individuals, shorter sleep duration, later sleep timing, and less sleep continuity are each associated with depressed mood and poorer subjective well-being (Merikanto et al., 2013; Totterdell, Reynolds, Parkinson, & Briner, 1994; Wong et al., 2013). These three sleep characteristics should thus be considered together in order to identify possible unique effects of each sleep component on mood.

Before investigating within-person variability in sleep characteristics, it is important to first recognize between-person variability, or individual differences, in these characteristics. Individuals can be characterized by their trait-like, average sleep characteristics. Studies often measure these individual differences through retrospective reports (e.g., “average sleep duration in the past month”) or behavioral (actigraphy) measures that are then averaged over a monitoring period (e.g., average sleep duration across 14 days). In the case of average sleep duration, studies show participants tend to sleep from <5 to >9 hours, with some individuals characterized as “short” sleepers and others “long” sleepers (Aeschbach et al., 2003; Grandner & Kripke, 2004). Similarly,
people differ in their average sleep continuity such that some are referred to as “good” and others “poor” sleepers (Buysse, Reynolds, Monk, Berman, & Kupfer, 1989). Lastly, people also naturally vary from one another in their sleep timing. The population distribution ranges from extreme “morning” to extreme” evening types, with some people’s sleep periods occurring much earlier and others much later than the population average, respectively (Roenneberg et al., 2004). Individual differences in each of these sleep characteristics have been linked to mood, with shorter sleep duration, poorer sleep continuity, and later sleep time associated with depressed mood and poorer well-being (Baglioni et al., 2011; Kaneita et al., 2006; Levandovski et al., 2011; Steptoe, O'Donnell, Marmot, & Wardle, 2008). An individual’s average sleep characteristics are thus important predictors of mood and overall well-being, and any effects of within-person fluctuations in sleep on mood are those that occur beyond the effects of baseline individual differences.

While studies often focus on people’s average sleep patterns, people exhibit variability in their sleep characteristics on a night-to-night basis, and effects of these within-person fluctuations are relatively understudied. While individuals with sleep disorders exhibit greater night-to-night variability than those without sleep disorders (Buysse et al., 2010), even healthy adults without sleep difficulties exhibit considerable variability (Buysse et al., 2010; Knutson, Rathouz, Yan, Liu, & Lauderdale, 2007). For instance, one study found that healthy adults without insomnia deviated on average by 53.9 min in their sleep duration (avg 6.63 hrs), 19.3 min in time spent awake at night (a measure of sleep continuity; avg 47.2 min), and 70.9 minutes in their reported bedtime (avg 11:26PM) from night-to-night (Buysse et al., 2010). These findings show that individuals exhibit fluctuations in their sleep characteristics on a day-to-day basis. Understanding the factors that regulate sleep will help elucidate possible mechanisms that drive these daily within-person variations in sleep and their covariation with mood.
There are several intrinsic factors that regulate sleep. First, the sleep cycle is primarily regulated by an interaction between the circadian system and homeostatic drive (Borbely, 1982; Dijk, Duffy, & Czeisler, 2000). The circadian system signals for wakefulness according to the 24-hour light/dark cycle, while the homeostatic drive refers to sleep need that increases with wakefulness (i.e., sleep debt). Specifically, sleep onsets when there is both a peak in homeostatic drive and a decrease in alertness, the latter of which is regulated by the circadian clock. Second, arousal is another factor that can influence people’s sleep patterns. Specifically, physiological or cognitive forms of arousal can delay the timing of sleep and lead to shorter sleep duration and/or poorer sleep continuity (Riemann et al., 2010; Tang & Harvey, 2004; Zoccola et al., 2009). An individual’s sleep characteristics can thus vary from day to day due to the interaction of these intrinsic factors. For example, an individual may experience poor sleep continuity and short sleep duration one night due to arousal states, which in turn leads to a buildup of the homeostatic drive (sleep debt) that can lead the individual to sleep at an earlier time and for a longer duration the following night. Taken together, sleep is a dynamic process and behavioral sleep characteristics on a given night can reflect both the individual’s experiences during the day and sleep from prior nights.

People can also experience nightly variability in their sleep characteristics due to environmental or social factors. First, light exposure has both a direct alerting effect on humans (Lockley et al., 2006) and is the most important cue for the circadian clock (Diane B Boivin, Duffy, Kronauer, & Czeisler, 1996). Aside from its role in entraining the circadian system, evidence shows that the use of light-emitting devises before bed associates with shorter sleep duration, more sleep disturbances, and a shift in the timing of sleep (Chang, Aeschbach, Duffy, & Czeisler, 2015; Fossum, Nordnes, Storemark, Bjorvatn, & Pallesen, 2014; Hysing et al., 2015). Prolonged
exposure to artificial lighting on a given day can thus alter an individual’s sleep characteristics. A second extrinsic factor involves social obligations and experiences. Shifts in work schedules and social events can dictate when an individual sleeps and wakes, even if at times not aligned with their circadian system (Ehlers, Frank, & Kupfer, 1988). Studies also show that exposure to psychosocial stressors, such as work stress or social conflicts, associates with shortened sleep duration, poor sleep quality, and more sleep disturbances (Åkerstedt et al., 2002; Knudsen, Ducharme, & Roman, 2007). Thus, while sleep is an intrinsically-regulated process, sleep characteristics may change as a function of varying social and environmental factors.

Taken together, people exhibit day-to-day variability in their sleep characteristics and there are numerous intrinsic and extrinsic factors driving these fluctuations. While the circadian and homeostatic processes are the primary regulators of sleep, exposure to varying environmental factors, social experiences, and arousal states on a given day can affect an individual’s sleep characteristics. If the sleep-mood association exists on a proximal, day-to-day basis, it would be expected that these within-person fluctuations in sleep characteristics would lead to covarying fluctuations in mood and vice versa.

1.1.2 Mood and Affect

Mood is a transient, long-lasting state that is comprised of feeling states known as affect (Watson, 2000). Individuals experience affect, an array of feeling states, throughout the day. Affect includes core emotions (i.e., sadness, joy, surprise, anger, fear, etc.), which are brief, intense feeling states that are high in activation (Watson, 2000). However, affect also includes feeling states that are not core emotions. Studies show that when participants are asked to indicate their feeling state and its intensity at multiple time points throughout the day, they more frequently
report mild-to-moderate feeling states that, unlike core emotions, can be diffuse, long lasting, and/or low in activation (e.g., fatigue and alertness; Watson, 2000). Individuals also tend to report experiencing more than one affect at a given time, and collectively, these feeling states comprise an individual’s mood (Watson, 2000). For instance, depressed mood can last throughout the day and include feelings of sadness, fatigue, and irritability. In other words, the term “affect” refers to a wide array of feeling states that comprise mood, and one approach to study how sleep relates to mood is to test the relationships between different sleep characteristics and types of affect.

Similar to sleep, affect can be further characterized by its dimensions. Studies show that self-reported affective states constitute two higher-order factors: positive affect (PA) and negative affect (NA) (Mayer & Gaschke, 1988; Tellegen, Watson, & Clark, 1999; Watson, Clark, & Tellegen, 1988; Watson & Tellegen, 1985). PA reflects one’s overall level of pleasurable engagement with the environment, with high PA including affective states such as active, joyful, delighted, and low PA including tired, down, fatigued (Watson & Tellegen, 1985). On the other hand, NA represents subjective distress. High NA involves negative states such as fearful, distressed, and angry, while low NA involves states such as relaxed and calm (Watson & Tellegen, 1985).

Of note, there is a long-debated issue in the literature concerning the independence of PA and NA, with some researchers arguing that PA and NA form a single bipolar construct rather than two independent factors (Barrett & Russell, 1999; Diener & Emmons, 1985; Russell & Carroll, 1999). This issue is relevant here because if PA and NA are bipolar ends of the same construct, then any reported association between sleep characteristics and PA may merely reflect the absence of NA or vice versa. In contrast, if the two factors were independent, then understanding how sleep characteristics relate to these two constructs would each have different implications (e.g., the
presence of positive feelings affects sleep, or sleep affects the presence of negative feelings). There is evidence that the negative correlation of PA and NA varies as a function of the temporal frame over which mood is assessed (Diener & Emmons, 1985). Specifically, when assessed in short time frames (e.g., in the past day, in the moment) PA and NA tend to covary inversely, such that when people report high levels of NA, they are not likely to simultaneously report high levels of PA. In contrast, when mood reports reference longer spans of time or are assessed at a global level, the correlation of PA and NA can be weak or negligible. Moreover, the PA-NA correlations also vary as a function of the assessment instrument (Egloff, 1998). For instance, one of the most common mood questionnaires, the Positive and Negative Affect Schedule (PANAS), was designed specifically to measure PA and NA as statistically independent factors (Crawford & Henry, 2004; Watson et al., 1988), and evidence shows a weak PA-NA correlation as measured by the PANAS, regardless of time frame (e.g., r’s: past week =-.14, in the moment =-.06) (Watson & Clark, 1997). Because the studies we will review used the PANAS, selected items from the PANAS, or derived orthogonal factors of PA and NA from other measures, results will be interpreted as pertaining to two independent, or weakly correlated dimensions of affect. Further descriptions of relevant assessment measures will be presented in Section 1.2.1.

Importantly, affect can be characterized at both a trait and state level. Trait affect refers to stable individual differences in the propensity to experience certain feeling states, and it is often measured through questionnaires asking participants to report how they “typically” feel or as an average of affective states recorded over multiple measurements. Although people differ from one another in their average affect, it is important to note that people exhibit dynamic changes in PA and NA over time (Eid & Diener, 1999; Penner, Shiffman, Paty, & Fritzsche, 1994; Stawski, Sliwinski, Almeida, & Smyth, 2008). These within-person fluctuations in affect can occur
moment-to-moment and day-to-day, and can be measured through multiple, repeated measures over the course of a given monitoring period. Each time point of measure assesses the person’s state affect, or how s/he felt short-term (e.g., in the past day, in the past 30 minutes). If there are proximal effects of affect on sleep patterns, or vice versa, it would be expected that these changes in state affect would associate with nightly sleep characteristics and that these effects occur beyond the effects of individual differences in trait affect. Understanding intrinsic and extrinsic factors that regulate within-person fluctuations in affect can help inform how affect covaries with sleep on a day-to-day basis.

One intrinsic factor that regulates affect is the circadian system. Healthy individuals show a diurnal rhythm in PA, characterized by a quadratic temporal pattern in which PA is initially low upon awakening, rises and peaks during the day, and declines throughout the evening (Lee Anna Clark, Watson, & Leeka, 1989; Murray, Allen, & Trinder, 2002). Evidence shows that these fluctuations are primarily regulated by the endogenous circadian system and the temporal pattern of PA is closely associated with that of core body temperature, another circadian rhythm (D. B. Boivin et al., 1997). While most studies report a diurnal rhythm in PA but not NA (e.g., Clark, Watson, & Leeka, 1989; Murray, Allen, & Trinder, 2002), there is some evidence that NA may also exhibit a diurnal rhythm (Miller et al., 2015; Peeters, Berkhof, Delespaul, Rottenberg, & Nicolson, 2006). Taken together, people exhibit regularly-occurring fluctuations in their levels of PA, and possibly in NA, throughout the day as a function of their intrinsic circadian system.

There are other extrinsic, environmental factors that can influence levels of affect, including that of daily psychosocial experiences. People exhibit affect reactivity, or changes in affect, in response to experiences such as work stressors, social conflicts, and positive social interactions (Mroczek & Almeida, 2004; Sliwinski, Almeida, Smyth, & Stawski, 2009; Stawski et
al., 2008). For instance, people tend to report more NA following exposure to stressors, and more PA following pleasant social experiences (McIntyre, Watson, Clark, & Cross, 1991; McIntyre, Watson, & Cunningham, 1990; Sliwinski et al., 2009). Details regarding this process will be reviewed in more depth in section 1.3 on Affect Reactivity.

Taken together, people exhibit day-to-day and moment-to-moment variability in their PA and NA. The circadian system partly drives this variability, with PA, and possibly NA, exhibiting a diurnal rhythm. Aside from this intrinsic factor, extrinsic factors such as varying daily experiences can influence momentary affect levels. If there is bidirectional association between sleep and affect, possible mechanisms that drive this relationship may involve these regulatory factors.

1.1.3 The Relationship between Sleep and Affect: A Bidirectional Model

Individuals show day-to-day variability in both their sleep patterns and their levels of affect, the latter of which can also vary from moment-to-moment. There are various intrinsic and extrinsic, environmental factors that regulate sleep and/or mood. Consideration of these factors together sheds light on possible mechanisms that can drive a proximal (day-to-day), bidirectional interplay between sleep and mood.

One mechanism through which sleep can alter mood is by altering processes that underlie how people emotionally respond to environmental factors. Experimental work shows that sleep deprivation can exaggerate how people perceive and respond emotionally to laboratory stimuli of negative and positive emotional valence (Franzen, Buysse, Dahl, Thompson, & Siegle, 2009; Gujar et al., 2011; Tempesta et al., 2010). Sleep deprivation may thus impact emotion regulation, a set of cognitive and neural processes that constitute people’s perception of and emotional
reactivity to emotionally evocative information (Gross & John, 2003; John & Gross, 2004). If emotion regulation mediates the sleep-affect relationship, it is possible that changes in sleep characteristics will influence not only time-averaged measures of affect but also people’s affect reactivity to psychosocial events. The extant literature will be reviewed in Section 1.3 on Affect Reactivity.

Changes in mood may lead to changes in sleep characteristics in part via arousal states. As noted earlier, both physiological and cognitive forms of so-called hyperarousal can regulate or disturb sleep. Specifically, excessive physiological arousal, such as hyperactivity in various neuroendocrine and neural systems, can delay the timing of sleep and lead to shorter sleep duration and/or poorer sleep continuity (Riemann et al., 2010). Studies have also shown that cognitive arousal during the day, in the form of rumination and worry, lead to shorter sleep duration and poorer continuity (Tang & Harvey, 2004; Zoccola et al., 2009). Because hyperarousal can stem from stress, anxiety, or depressed mood, the effect of arousal on sleep may be one mechanism through which mood states affect sleep characteristics.

Given that sleep and mood are often associated, and people exhibit within-person variation in both, we hypothesize that the sleep-mood association is bidirectional and occurs on a proximal, day-to-day basis. We propose a model that incorporates several plausible mechanisms (Figure 1). First, acute changes in sleep characteristics may alter individuals’ abilities to process and regulate their emotional responses to environmental factors, which may be reflected in their reported affective responses to events or provocations experienced the following day. Subsequent changes in mood may in turn occasion states of hyperarousal, which can then change an individual’s sleep characteristics that corresponding night.
The current study posits that (A) changes in sleep characteristics predict changes in next day levels of affect, which then lead to changes in nighttime sleep characteristics, and (B) changes in sleep characteristics predict changes in affect reactivity to daily experiences.

Although not depicted in the model (Figure 1), there are other pathways that may underlie the relationships between sleep and mood. As noted above, altered emotion regulation is one pathway through which sleep disruptions might lead to depressed mood. However, impaired emotion regulation that occurs with depressed mood could in turn lead to greater hyperarousal at night and disturbed sleep. In other words, this mechanism might likewise underlie the influence of mood on sleep. Other forms of reactivity, such as physiological reactivity, may also be involved in the sleep-mood relationship. For instance, there is evidence that sleep disruptions increase blood pressure reactivity to stressors (Franzen et al., 2011) and greater blood pressure reactivity has been
related to depression (Ayduk & Kross, 2008). Physiological changes in the cardiovascular system that occur after sleep disruptions may in part mediate the effects of sleep on mood. In addition, psychological stress has been shown to increase physiological arousal during sleep (M. Hall et al., 2007), which suggests that daytime experiences can have direct effects on sleep physiology. Taken together, there are alternate mechanisms that are important to consider when interpreting any findings regarding the sleep-affect relationship.

Other intrinsic factors that regulate sleep and mood need to be considered as well. As described earlier, an individual’s sleep characteristics on a given night can reflect their sleep experiences from previous nights (e.g., sleep debt) and the influence of extrinsic factors such as work schedules (e.g., obligated wake times). In our model, we propose there are effects of mood on sleep characteristics that occur even after accounting for these factors. In addition, because an individual’s mood is regulated by the circadian system, individuals exhibit fluctuations in mood according to the time of day. We propose that the effects of sleep characteristics on affect reactivity are independent of this underlying diurnal rhythm in affect.

In sum, we propose that sleep characteristics on a given night predict levels of PA and NA the following day, while affect on a given day will predict sleep characteristics that night. In addition, we hypothesize that changes in sleep characteristics will associate with changes in affect reactivity. The following literature review will 1) evaluate existing evidence regarding any temporal associations between sleep and mood, and 2) discuss what is known and not known regarding the effects of sleep characteristics on affect reactivity.
1.2 Effects of Sleep on Affect: Experimental Evidence

Experimental studies have tested the sleep-affect relationship by examining participants’ change in affect after altering their sleep characteristics experimentally. Such manipulations include total sleep deprivation and partial sleep restriction, both of which allow researchers to test the effects of both complete sleep loss and changes in sleep duration. In addition, researchers have tested the effects of poor sleep continuity on affect, independent of the effects of sleep duration, by inducing frequent awakenings in participants while preserving their overall sleep duration. Lastly, researchers have tested for changes in affect following shifts in participants’ sleep time (bedtime, wake time) and after sleep times are kept consistent, again while maintaining consistent sleep duration. In all studies, participants were instructed to maintain consistent sleep schedules that allowed for ample sleep opportunity across several days prior to sleeping in the laboratory, which helped prevent potential lag effects of previous sleep patterns (e.g., sleep debt, shifts in sleep time, etc.). In other words, results from these studies provide evidence regarding how an individual’s affect directly covaries with changes in their sleep characteristics.

Overall, studies have found significant effects of sleep loss on affect. Following total sleep deprivation, participants have reported emotional distress or increased NA and reduced PA, relative to the day prior to deprivation (Babson, Trainor, Feldner, & Blumenthal, 2010; Baum et al., 2014; Franzen et al., 2008; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). While sleep deprivation studies represent extreme cases of sleep loss, sleep restriction studies have aimed to test the effect of moderate sleep restriction (4-5 hrs) that people more commonly experience. Sleep restriction has been shown to induce changes in PA and NA similar to total sleep deprivation and after as little as one night of sleep restriction (Baum et al., 2014; Dinges et al., 1997; Haack & Mullington, 2005; Kahn et al., 2014). If these findings generalize to naturally-occurring, day-to-
day variability in sleep duration, individuals encountering even one night of curtailed sleep may experience reduced PA and elevated NA relative to their average levels of affect.

Like sleep restriction, poor sleep continuity in the form of frequent awakenings can lead to changes in affect (Stepanski, 2002). Studies have shown that after participants were frequently awoken (once every minute of sleep), they reported lower PA and feeling more “unhappy” the next day (Bonnet, Berry, & Arand, 1991; M. H. Bonnet, 1985). Of note, these early studies tested the effects of imposing very frequent, brief awakenings in an attempt to mirror the nighttime awakenings experienced by patients with breathing-related sleep disorders. A more recent study aimed to test whether longer lasting and relatively less frequent awakenings akin to those experienced by the general population would similarly influence affect (Kahn et al., 2014). Participants slept at home and were woken via telephone call by research assistants every 90 minutes (4x total) and asked to stay awake for 15 minutes each time. After experiencing a night of this poor sleep continuity, the participants reported greater NA and emotional distress the next day. These findings suggest that poor sleep continuity increases NA and decreases PA, and that these effects can be observed following a single night of disrupted sleep.

In comparison to sleep duration and continuity, fewer laboratory studies have investigated the effects of sleep timing on affect. Existing studies have shown that when healthy adults were asked to sleep 2-4 hours earlier or later than usual, they reported more NA and less PA the next day (Taub & Berger, 1974, 1976). In other words, changes in the form of either advances or delays in sleep timing appear to influence affect. Of note, in both studies, participants’ sleep duration did not differ between control and shift conditions. Findings thus suggest that sleep timing is an understudied sleep characteristic that may have proximal effects on mood.
Overall, experimental studies show that short sleep duration, poor sleep continuity, and shifts in sleep timing, can increase NA and decrease PA. These findings are consistent with population studies that show short sleep duration and sleep disturbances associated with greater NA and less PA (Bower, Bylsma, Morris, & Rottenberg, 2010; Fuligni & Hardway, 2006; Steptoe et al., 2008). In regard to sleep timing, there is a large literature linking evening chronotype (preference for late sleep time) to less PA and more depressed mood (Biss & Hasher, 2012; Hasler, Allen, Sbarra, Bootzin, & Bernert, 2010; Hasler et al., 2012; Hidalgo et al., 2009; Levandovski et al., 2011). Experimental findings suggest that changes to an individual’s sleep timing, including both advances and delays, rather than late sleep timing itself, can lead to changes in affect. Taken together, experimental work extends upon findings from epidemiological studies and shows that within-person changes in sleep characteristics can lead to changes in affect observable the next day.

It is important to note several limitations in this literature. First, these studies are based on brief, artificially-induced sleep patterns. Second, these studies are designed solely to examine the effects of sleep changes on affect and therefore are uninformative regarding influences of affect on sleep, hence not addressing the potential for a bidirectional relationship. Another set of studies that complements this experimental work addresses the relationship between day-to-day changes in sleep characteristics and affect within naturalistic settings. If the effects of sleep manipulations generalize to naturally-occurring sleep characteristics, it is predicted that after nights when individuals sleep less, have poorer continuity, or sleep at times that deviate from their average sleep patterns, they will report more NA and less PA. If this relationship is bidirectional, it is predicted that on days when individuals report more NA and less PA compared to their average
levels of affect, they will sleep less, have poorer sleep continuity, and sleep at times that deviate from their average sleep patterns.

1.3 The Temporal Relationship Between Sleep and Affect

In addition to the studies regarding effects of manipulated sleep on affect reviewed above, observational studies have begun to elucidate day-to-day relationships between naturally-occurring sleep characteristics and affect. While findings in this literature begin to support a bidirectional model, results are mixed (see Tables 1 and 2) and may reflect methodological differences among studies. Briefly, some studies show that after individuals sleep relatively shorter durations or have less sleep continuity compared to their average patterns, they subsequently experience relatively less PA (de Wild-Hartmann et al., 2013; Mccrae et al., 2008; Scott & Judge, 2006; Sonnentag, Binnewies, & Mojza, 2008; Totterdell et al., 1994; Wrzus, Wagner, & Riediger, 2014) or more NA the following day (Brissette & Cohen, 2002; de Wild-Hartmann et al., 2013; Galambos, Dalton, & Maggs, 2009; Mccrae et al., 2008; Scott & Judge, 2006; Wrzus et al., 2014). Some findings show that when individuals report greater NA or less PA than their average on a given day, they subsequently experience shorter sleep duration and less sleep continuity that night (Brissette & Cohen, 2002; Kalmbach, Pillai, Roth, & Drake, 2014). In regard to sleep timing, the one study of healthy adults found later sleep onset to predict lower next-day PA (Totterdelle et al., 1994), but no studies have examined the effect of sleep timing on NA or the influence of daytime affect on sleep timing. Taken together, the relationships between these sleep characteristics and affect remain unclear, either due to inconsistent evidence or in the case of sleep timing, due to a
paucity of studies. Understanding the design and methodology of these studies will provide scaffolding for further interpretation of results.

1.3.1 Study Designs, Methodology, and Participant Demographics

Before examining study results, the following section will describe factors that may contribute to inconsistent findings, including participant demographics, study design, statistical framework, and the various tools for assessing sleep characteristics and affect that are used across studies. Here, differences in each of these study aspects will be briefly noted but the limitations and implications of each method will be explored in more depth later in the interpretation of study results.

1.3.1.1 Participant Demographics

While there is literature on the sleep-affect association in clinical cohorts, such as chronic pain patients and persons with mood disorders, the use of medications and comorbidity of other symptoms may limit generalizability of findings. Consideration of this literature is thus beyond the scope of the current study. In addition, sleep characteristics, mood and affect levels change with age, such that older age is associated with shorter sleep duration, earlier sleep timing, less sleep continuity, and less NA and more PA

(Brabbins et al., 1993; Charles, Reynolds, & Gatz, 2001; Ohayon & Vecchierini, 2005; Reyner & Horne, 1995; Roenneberg et al., 2004; Unruh et al., 2008). Results from studies that exclusively examine children, adolescents (<18 years old), or older adults (>65 years old) may thus be specific to those developmental stages and not generalizable. Based on these considerations, the studies that will be further examined are those on healthy adults. Participants
across studies were similar in demographic characteristics and were predominantly highly educated, White Caucasian adults. Thus, any inconsistent results across studies reviewed here are not likely attributable to differences in sample demographics.

1.3.1.2 Study Design and Statistical Framework

All the studies considered here implemented a prospective study design and a hierarchical linear modeling framework. Specifically, the studies assessed participants’ sleep characteristics and levels of affect on a daily basis, with time frames ranging from 5 to 21 consecutive days. Such study designs result in nested, hierarchically organized data. At the lower level, repeated measures of sleep and affect are nested within each participant (i.e., sleep characteristics, PA, and NA collected per participant, per day). At the next level are data regarding individual differences (between-person variables), including the participant’s demographics, and their average sleep characteristics and average PA and NA. In order to analyze these data, each of the studies considered here implemented hierarchical linear modeling (HLM), also known as random effects, multilevel, or mixed modeling. HLM incorporates this nested data framework and allows for the study of within-person changes over time while considering possible differences between persons that may confound the outcomes of interest (N Bolger & Laurenceau, 2013). For example, by using this study design and statistical framework, researchers can examine how day-to-day deviations in an individual’s sleep duration from his/her average sleep duration may relate to deviations in his/her affect. In other words, this framework allows researchers to control for between-person differences in baseline PA and NA levels or in sleep characteristics. Across each of the studies considered here, the researchers tested whether individuals’ sleep characteristics on a given night predicted PA or NA levels the following day and/or whether affect levels on a given day predicted sleep characteristics that night.
The studies considered here are comparable in study design and general analytical framework. However, studies differed in model covariates. For instance, Kalmbach et al. (2014) included measures of participants’ previous night sleep characteristics and previous day affect levels as covariates in the model. This comprehensive approach takes into account the possibility that on a given day, any observed relationship between an individual’s affect and sleep characteristics is explained by his/her sleep and affect experiences from the day prior. For instance, longer sleep duration on a given night may not be directly caused by changes in the individual’s affect levels but rather by a culmination of sleep debt from consecutive nights of short sleep. Inclusion of previous day measures as control variables is thus important to infer relationships of directionality. However, the majority of studies did not consider such lag effects. Differences in statistical models and their implications will be further discussed when interpreting study results.

1.3.1.3 Sleep Assessments

Various approaches can be used to characterize an individual’s sleep patterns, and differences in sleep assessment can influence study findings. Polysomnography (PSG) involves a multi-parametric, comprehensive monitoring of various biophysiological processes, including brain activity, eye movement, rate of breathing, and heart rate. This method is often referred to as the gold-standard measure of sleep and provides information regarding an individual’s sleep characteristics while the participants sleep in controlled laboratory settings. Another assessment tool is actigraphy, the use of watch-like, accelerometer devices (Sadeh & Acebo, 2002). Using this method, researchers estimate participants’ sleep characteristics based on their activity levels and periods of rest (i.e., lack of activity). Because actigraphy devices are ambulatory, researchers can use this method to monitor participants in their home environments over an extended period (e.g., 2 weeks). Lastly, researchers can quantify sleep characteristics based on participants’ reports
through sleep diaries, questionnaires, or phone interviews. Using this method, researchers ask participants to report their perceived sleep characteristics for a given time period (e.g., the previous night, or their average sleep characteristics over the past week, month, or year). Taken together, participants’ sleep patterns can be characterized physiologically through PSG, behaviorally through actigraphy, and subjectively through various self-report tools.

Studies that are aimed at assessing participants’ habitual sleep characteristics in their, home environments often rely on self-report methods. As shown in Tables 1 and 2, each of the studies quantified sleep characteristics based on retrospective reports that occurred once a day, either in the morning or evening. At the time of assessment, participants were asked to report their sleep characteristics for the previous night. The majority of studies created interviews or sleep logs to assess sleep patterns. Two studies used select item(s) from the Pittsburgh Sleep Quality Index (PSQI; Buysse et al., 1989), a validated questionnaire used to assess subjective sleep quality and disturbances, with questions modified to reflect daily sleep. Another study used a modified version of the Jenkins insomnia scale (Jenkins, Stanton, Niemcryk, & Rose, 1988), which asks participants to rate descriptions of sleep continuity (e.g., had trouble falling asleep, woke up several times at night, etc.). With the exception of the study that used the Jenkins scale and its aggregate sleep continuity score (Scott & Judge, 2002), all studies similarly quantified each sleep characteristic (duration, continuity, timing) based on a single item.

Self-reported sleep characteristics are often only minimally correlated with those quantified by PSG, with significant differences between sleep diary and PSG-derived sleep duration and sleep continuity (Kushida et al., 2001; McCall & McCall, 2012; Silva et al., 2007). For instance, in a large study on healthy adults, Silva et al. (2007) compared participants’ morning reports of their sleep characteristics the previous night to corresponding measures derived from
PSG. Both total sleep time (sleep duration) and the time it took to fall asleep (a measure of continuity) were minimally correlated with PSG (r’s=.14-.16), with participants’ self-reports being longer relative to PSG. That there is a large degree of unshared variance between PSG-derived and self-reported sleep characteristics suggests that subjective experiences may reflect recall bias and do not necessarily correspond with objectively determined sleep patterns.

Aside from self-report, actigraphy is often used to quantify participant sleep patterns outside of the laboratory. Actigraphy has been validated against PSG, and the epoch-by-epoch agreement rates between the two methods in detecting sleep are high, particularly for healthy individuals (> .85 agreement rates; Sadeh & Acebo, 2002; (Ancoli-Israel et al., 2003; Jean-Louis, Kripke, Cole, Assmus, & Langer, 2001; Marino et al., 2013). The correlations between self-reported sleep characteristics and those from actigraphy are uniformly higher than with respect to PSG, albeit not strong. Several studies on adult participants have shown moderate correlations between an individual’s self-reported and actigraphy-derived measure of sleep duration, with most participants tending to overestimate their own sleep duration in comparison to actigraphy (r’s=.34-.57; Auger, Varghese, Silber, & Slocumb, 2013; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008; Lockley, Skene, & Arendt, 1999; McCall & McCall, 2012; Tomita et al., 2013). One study estimated that among healthy adults, 34% of participants reported sleep durations that deviated ±1 hour from actigraphy-derived duration, with most of self-report durations being longer than the actigraphy measure (Van Den Berg et al., 2008). Studies have shown moderate to high correlations between actigraphy and self-reported sleep timing (onset and offset, r’s=.57-.77) and widely varying correlations for different forms of sleep continuity, such as awakenings and time it takes to fall asleep (r’s=.06-.59; Lockley et al., 1999; McCall & McCall, 2012). Of note, it is possible that reported correlations between self-report and actigraphy measures are overestimates because
researchers at times integrate self-report records into actigraphy data to reconcile ambiguous or missing actigraphy data. These findings suggest that actigraphy-derived sleep characteristics represent the behavioral aspect of sleep patterns that are correlated but distinct from corresponding subjective measures.

1.3.1.4 Measures of Affect

To examine the day-to-day relationship between sleep and affect, studies assessed affect on a daily basis, either via once-daily retrospective reports or through ecological momentary assessment (EMA). The self-report questionnaires and general EMA methods are briefly described below.

The majority of studies relied on retrospective, once-a-day reports, and specifically administered the Positive and Negative Affect Schedule (PANAS), the extended version (PANAS-X), or selected items from the questionnaire in a daily log. The PANAS is a 20-item scale on which participants rate descriptors on a Likert scale (1, very slight or not at all, to 5, extremely) based on how they felt during an instructed time interval (Watson et al., 1988). These studies asked participants to rate how they felt “today.” Of note, the PANAS was designed to assess the general dimensions of PA and NA, and was constructed to contain items that were statistically pure markers of either dimension (Watson et al., 1988). In other words, only items that loaded substantially on one factor and not the other were included in the questionnaire. As a result, by using the PANAS to measure affect, the researchers are examining affect as two orthogonal dimensions (PA and NA).

Two studies used the PANAS-X, an extended version of the original PANAS that includes 60 items (Watson & Clark, 1999). In addition to assessing the general dimensions of PA and NA, the PANAS-X also measures 11 specific affects (e.g., fear, sadness, joviality, attentiveness, guilt,
hostility, fatigue, surprise). Although the two studies reported the relationship between sleep characteristics and specific PANAS-X subscales (Scott & Judge, 2006; Kalmbach et al., 2014), results across studies will be discussed more generally in regard to PA or NA.

Aside from the PANAS, one study assessed level of negative mood with items taken from the Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1992; McNair, Lorr, & Droppleman, 1981). The POMS is a questionnaire designed to assess current mood states and contains 65 words or phrases that participants are asked to rate on a 5-point Likert scale (0, not at all, to 4, extremely) based on how they were feeling for two timeframes: the past week or currently (McNair et al., 1981). The resulting scores form 4 negative mood subscales (Tension, Depression, Anger, Fatigue, Confusion) and 1 positive mood subscale (Vigor). Unlike the PANAS, the POMS was designed to quantify overall negative mood rather than specific PA and NA dimensions. Of note, however, the POMS subscales are highly correlated with 5 corresponding scales from the PANAS-X (r = .85 to .91), including a high correlation between Vigor and the PA scale (r = .86; Watson & Clark, 1994). Here, the one study that administered the POMS (Brissette & Cohen, 2002) used only a portion of the questionnaire items and derived orthogonal PA and NA factors from the resulting data.

An alternative to retrospective report is the use of ecological momentary assessments (EMA). EMA is a method that uses repeated sampling strategies to assess a given phenomenon at or close to the moment that it occurs, while participants are in their natural setting (Stone & Shiffman, 1994). This approach aims to maximize ecological validity while avoiding recall bias that occurs with retrospective reports. For instance, when recalling their average affect for the day, people can be biased by the recency or salience of an emotional experience. Evidence suggests people are more likely to recall negative information and less easily recall positive information.
when they are in a negative mood at the time of assessment (Clark & Teasdale, 1982; Kihlstrom, Eich, Sandbrand, & Tobias, 2000). In other words, if an individual is in a negative mood when completing the affect assessment, they may overestimate the average level of NA they experienced throughout the day. With EMA, researchers can repeatedly sample participants’ affect at random or preset times (e.g., every two hours after awakening) and aggregate the data across different time frames of interest. Here, three of the studies used EMA methods to collect multiple samples of PA and NA throughout each day and calculated a daily PA score and NA score for each participant. Such an approach allows researchers to collect data on a momentary basis, which avoids potential recall bias, and to use these data to derive daily scores of participants’ average affect levels.

By using EMA, researchers are able to collect data at preset time schedules and also collect data regarding possible antecedents of affect changes. For instance, among the reviewed studies, three used EMA methods in which participants were prompted at fixed intervals after awakening (every 1.5-2 hrs) to complete a battery of affect items (Totterdell et al., 1994; de Wild-Hartmann et al., 2013; Kalmbach et al., 2014). By sampling across multiple time intervals, researchers are able to control for time-of-day effects, which provides another advantage over once-a-day reports given the diurnal rhythm of affect (Clark et al., 1989; Murray et al., 2002). In addition, EMA can be used to collect data on environmental factors that covary with affect (Shiffman, Stone, & Hufford, 2008). For instance, because EMA elicits information on a momentary basis, it is possible to relate factors (e.g., i.e., work stressors) to changes in affect. In other words, the EMA method not only improves upon the limitations of once-a-day reports, but also allows researchers to study dynamic changes in affect over time and in relation to other environmental or experiential factors. However, the studies reviewed here only focused on participants’ average daily affect levels rather than changes in affect throughout the day. The use of EMA to examine how sleep characteristics
influence affect changes over time and in response to situational factors will be explored in Section
1.3 (Affect Reactivity).

In the next sections, results across studies will be discussed in relation to the reported sleep
characteristic (duration, continuity, or timing) and measure of affect (PA or NA) and interpreted
with respect to methodology and findings from experimental and population studies.

1.3.2 The Day-to-Day Effects of Sleep Characteristics on Affect

1.3.2.1 The Effect of Sleep Duration on PA and NA

As shown in Table 1, 7 studies tested whether sleep duration on a given night predicted PA the following day. Of these studies, 4 reported null findings (Totterdell et al., 1994; Brisette & Cohen, 2002; Galambos et al., 2009; Kalmbach et al., 2014). Three studies reported significant but different effects. Two found that when individuals slept less relative to their average length, they reported lower than their average PA the following day (Sonnentag et al. 2008; de Wild-Hartmann et al., 2013). On the other hand, Wrzus et al. (2014) found that when individuals slept either less or more than their average sleep duration, they reported lower PA. All significant effects were small to moderate in size.

In regard to NA, six studies examined whether sleep duration on a given night predicted NA the following day. Three of these reported no associations (Sonnentag et al., 2008; de Wild-Hartmann et al., 2013; Kalmbach et al., 2014) and three reported significant, but different effects. Two of the latter studies showed a negative, linear relationship between sleep duration and NA, such that when individuals slept less than their average sleep duration, they reported more NA the following day (Brissette & Cohen, 2002; Galambos et al., 2009), and in the third, Wrzus et al. (2014) again reported a nonlinear relationship such that when individuals slept either less or more
than their average sleep duration, they reported more NA the following day (Wrzus et al., 2014). All significant effects were small to moderate in size.

In sum, half of available studies reported significant effects of sleep duration on affect, with effect sizes ranging from small to moderate. Of note, none of the studies reported the within-person standard deviations in sleep duration or in PA and NA, so that it is unknown whether differences in variability of sleep duration and affect may distinguish studies that found significant results and those that did not. On the other hand, there were no systematic differences in study design or assessment tools between studies that reported small effects, medium effects, or null findings. Still, there are notable methodological limitations in this literature, including 1) reliance on self-report sleep assessments, 2) the lack of appropriate control variables in each model, and 3) once-a-day assessments of affect. Interpretation of study results in the context of other literature, such as experimental sleep studies, points to the potential influence of variation in study design and methodology and is discussed further below.

One methodological factor that may contribute to inconsistencies is the method of sleep assessment. The reviewed studies all assessed sleep duration through subjective reports. Since the experimental studies cited earlier manipulated participants’ sleep duration and measured sleep by either actigraphy or PSG, it is possible that the discrepancy between findings from these studies and those from experimental work are partly due to type of sleep assessment. As discussed earlier, self-reported sleep duration does not strongly corroborate behaviorally or physiologically determined sleep duration, with only minimal or moderate correlation between subjective reports and corresponding actigraphy and PSG measures. Thus, self-reported restrictions in sleep duration may not mirror the objectively-determined sleep restriction in experimental studies.
Aside from sleep assessment methods, night-to-night variability in sleep duration may influence study results. On average, participants in these naturalistic studies reported sleeping between 7-8 hours. However, none of the studies reported within-person standard deviations in sleep duration. Thus, it is unknown if there were differences in sleep duration variability between studies that found significant results and those that reported null findings. In experimental sleep restriction studies, participants were restricted from their habitual, 7-9 hr sleep duration to 4-5 hrs per night (Dinges et al., 1997; Haack & Mullington, 2005; Kahn et al., 2014; Baum et al., 2014). It is possible that the observed effects of sleep restriction on affect occur only when individuals experience a relatively large deviation in sleep duration (or large amount of sleep restriction). Studies of healthy adults have found that individuals experience approximately 1-hour deviations from night to night in their sleep duration (Knutson et al., 2007; Buysse et al., 2010). It is thus possible that null effects occurred in part because participants did not naturally experience sufficient variation in sleep duration to influence next-day affect in a manner comparable to experimental studies.

Another methodological limitation concerns the assessment of affect. Most of the reviewed studies evaluated PA and NA through self-report measures taken once a day. As discussed earlier, these one-time measures of affect can be confounded by recall bias. In contrast, the use of EMA allows researchers to collect repeated measures of affect throughout the day, which avoids recall bias and potential time-of-day influences. Here, three of the studies used EMA methods to derive daily PA and NA scores (Totterdell et al., 1994; de Wild-Hartmann et al., 2013; Wrzus et al., 2014), but reported differing results. And it may be noted that, to date, no studies have examined the day-to-day relationship between sleep and affect while using both EMA measures of affect and instrumented sleep assessments.
Most of the reviewed studies also do not consider potential lagged effects of affect and sleep characteristics. As discussed earlier, because these prospective studies aim to infer proximal (day-to-day) effects of sleep duration on affect, it is important to consider the potential lag effects of affect and sleep patterns from prior days. For instance, if a participant sleeps less on Night 1 and reports lower PA on Day 2, it is possible that the individual’s sleep duration directly influenced his/her affect. Or, it is possible that the participant reported a low level of PA on Day 1, which predicted short sleep duration on Night 1 as well as low PA on Day 2. Inclusion of lag variables (e.g., PA_{day-1}) is necessary to infer directionality. Only two of the reviewed studies controlled for the lag effects of previous day affect, and neither found an effect of sleep duration on reported affect (Totterdell et al., 1994; Kalmbach et al., 2014). It is possible that effects of sleep duration observed in other studies are in fact masking lag effects of affect. More research is needed to examine these associations while appropriately including lag variables in the statistical model.

Aside from lag effects, future studies can extend the current literature by considering the cumulative effects of sleep duration. Evidence from experimental literature suggests that sleep duration not only has an immediate impact on affect, but that these effects can cumulate. For instance, two studies that measured daily affect over several consecutive days of sleep restriction reported a cumulative effect of short sleep duration (i.e, 4-5 hours/night), wherein participants’ overall negative mood continued to increase and PA continued to decrease across days of restricted sleep (Dinges et al., 1997; Haack & Mullington, 2005). Studies are warranted to replicate these findings in naturalistic settings and to test whether individuals experience an increasing level of NA and decreasing level of PA after sleeping less on consecutive nights.

Another approach to expand upon the statistical model is to consider non-linear associations between sleep duration and affect. Aside from one study, all others only tested for
linear effects of sleep duration. In contrast, Wrzus et al. (2014) reported testing for both a linear and non-linear relationship between sleep duration and affect. The authors also found that the effect of sleep duration on affect depended upon participant age. After categorizing the participants (12-88 years old) by age, the authors found that adolescents and younger adults showed a linear relationship between sleep duration and affect (e.g., shorter sleep duration predicted less PA and more NA). In contrast, participants 20 years and older exhibited a quadratic effect in which both shorter and longer sleep duration predicted less PA and more NA, suggesting that the influence of sleep duration on affect changes as a function of ages. While these preliminary findings suggest that age may modify the effects of sleep on affect and that there may be a non-linear association specifically in adults, most studies to date have not tested these possibilities.

In summary, existing prospective studies report mixed results, with only half of the studies showing sleep duration on a given night to predict PA, NA, or both the following day. A range of methodological and statistical limitations may contribute to these inconsistencies. First, studies have uniformly relied on retrospective reports of sleep characteristics and of affect (one report per day), which are subject to bias. In addition, studies have largely neglected to account for the potential lag effects of sleep and affect, which confounds interpretation of temporal relationships. Last, studies on adults have not yet considered whether there may be culminative or nonlinear effects of sleep duration on affect.

1.3.2.2 The Effect of Sleep Continuity on PA and NA

As previously described, sleep continuity refers to disruptions (or lack thereof) to an individual’s sleep period and takes into account the time it takes to fall asleep (sleep latency), awakenings, and time spent awake during the sleep period. For ease of interpretation, studies that assessed at least one form of these disruptions are included in this section.
Six studies tested whether less sleep continuity on a given night predicted PA the following day, four of which found that when individuals reported less sleep continuity on a given night than their average amount, they reported less PA the following day (Totterdell et al., 1994; Scott & Judge, 2002; McCrae et al., 2008; de Wild-Hartmann et al., 2013). The remaining two studies reported null effects (Brissette & Cohen, 2002; Kalmbach et al., 2014). In regard to NA, five studies tested whether sleep continuity on a given night predicted NA the following day. Similar to the studies on PA, the majority (four out of five studies) found that when individuals reported less than their average sleep continuity on a given night, they subsequently reported more than their average NA the following day (Brissette & Cohen, 2002; Scott & Judge, 2002; McCrae et al., 2008; de Wild-Hartmann et al., 2013;). And one study reported no significant effect of sleep continuity on NA (Kalmbach et al., 2014).

Most studies showed that poorer sleep continuity predicts more NA and less PA. There was no systematic difference in participant demographics or methodology between studies reporting positive or null effects. Of note, all but one study assessed sleep continuity through self-report measures. Interestingly, McCrae et al. (2008), who tested the effects of both self-reported and actigraphy-derived nighttime awakenings, found that perceived, but not objectively defined, time spent awake at night predicted affect the following day. These results contradict experimental evidence that induced awakenings on a given night lead to greater NA and less PA the next day (Bonnet 1985; Bonnet et al., 1991; Kahn et al., 2014). Of note, the average duration that participants were awake at night in the naturalistic study (53.4 min; McCrae et al., 2008) was comparable with that in experimental studies (e.g., 60.0 min; Kahn et al., 2014). However, McCrae et al. (2008) did not report within-person standard deviations in awake time. It is unknown if participants experienced a degree of variability in sleep continuity comparable to that in
experimental studies, and whether differences in this variability may have contributed to this null finding. The evidence that supports a link between sleep continuity and affect is solely based on self-report assessments of sleep, which brings to question whether the observed associations between sleep continuity and affect may be specific to perceptions of sleep.

When investigating the relationship between sleep continuity and affect it is also important to draw from literature on sleep quality. Sleep quality refers to an individual’s satisfaction with and perception of his/her sleep. Measures of sleep quality often incorporate or overlap with measures of sleep continuity. For example, the Pittsburgh Sleep Quality Index (PSQI) is a commonly used questionnaire that provides a global sleep quality score derived from items tapping perceived quality, as well as number of awakenings, time it takes to fall asleep, and other reports of sleep continuity (Buysse et al., 1989). In parallel with results drawn from measures of sleep continuity, an individual’s sleep quality on a given night predicts both PA and NA the following day. Five of the aforementioned studies included a measure of sleep quality. All of these studies found that when individuals reported lower sleep quality on a given night, they also reported lower PA the following day (Totterdell et al., 1997; McCrae et al., 2008; Sonnentag et al. 2008; de Wild-Hartmann et al. 2013; Kalmbach et al., 2014). In regard to NA, three of four studies found that when individuals reported lower sleep quality on a given night, they reported greater NA the following day (McCrae et al. 2008; Sonnentag et al. 2008; de Wild-Hartmann et al., 2013; Kalmbach et al., 2013).

Studies that have investigated either specific aspects of sleep continuity (e.g., awakenings) and sleep quality are consistent in showing an association between perceived sleep continuity and affect. Since participants’ sleep quality and self-reported continuity may be influenced by mood
state, though, it is unclear whether these findings generalize to objectively quantified sleep continuity.

1.3.2.3 The Effect of Sleep Timing on PA and NA

While there is a growing literature on the association between individual differences in sleep timing and mood, there is a paucity of studies on the within-person, direct effect of night-to-night shifts in sleep timing on affect. The one study that examined the effect of sleep timing on PA found that later sleep onset predicted less PA (cheerfulness) the following day (Totterdell et al., 1994). No study examined whether sleep timing influences NA.

Thus, preliminary findings suggest that within-person variability in sleep times can alter affect, at least in terms of PA. This finding is consistent with evidence that individuals who report late sleep timing or evening chronotype (which represents a preference for late sleep timing) also report lower levels of PA relative to those who report a preference for earlier sleep timing (Biss & Hasher, 2012; Hasler et al., 2010; Hasler et al., 2012). Although no studies to date have tested the effect of day-to-day sleep time variability on NA, population studies show that individuals who report late sleep timing also report more NA relative to those with earlier sleep timing (Hidalgo et al., 2009; Levandovski et al., 2011). In addition, experimental literature has shown that within-person shifts (both advances and delays) in sleep timing lead to increased NA and decreased PA (Taub & Berger 1974, 1976). In other words, it may be that variability in sleep timing, rather than sleep timing per se, alters affect. Based on collective findings from population and experimental literature, it is predicted that when individuals sleep at times earlier or later than their average sleep time, they will report relatively more NA and less PA the following day.
1.3.3 The Day to Day Impact of Affect on Sleep

While most research has focused on the effect of sleep characteristics on affect, several studies have examined whether the sleep-affect association is bidirectional and tested for the effect of daytime affect on sleep characteristics. These are summarized below.

1.3.3.1 The Effect of PA on Sleep Characteristics

Five studies tested whether an individual’s daytime level of PA influences his/her nighttime sleep characteristics (de Wild-Hartmann et al., 2013; Galambos et al., 2009; Kalmbach et al., 2014; Scott & Judge, 2006; Totterdell et al., 1994). Of these studies, only Kalmbach et al. (2014) reported a significant association, where on days participants reported higher PA, they experienced more sleep continuity and longer sleep duration that night. Notably, two studies found that while PA did not predict measures of sleep continuity (awakenings, difficulty falling asleep), there was a positive association between PA and perceived sleep quality (de Wild-Hartmann et al. 2013; Galambos et al., 2009). Only one study tested the effect of PA on sleep timing, finding no significant effect (Totterdell et al., 1994).

Existing studies suggest, with one exception (Kalmbach et al., 2014), that the association between sleep characteristics and PA is primarily unidirectional, with sleep characteristics predicting levels of PA. There were no systematic differences in samples or assessment tools between the one exception and studies that reported null effects. Notably, Kalmbach et al. (2014) included a more comprehensive statistical framework in comparison to other studies and controlled for participants’ previous day sleep and affect characteristics. It is thus unlikely that findings from this study were confounded by previous sleep or affective experiences. However, all studies were limited to retrospective reports of sleep characteristics and affect. Interestingly, two studies
showed that lower PA associated with poorer ratings of sleep quality but not specific aspects of sleep continuity. Taken together, preliminary findings suggest that fluctuations in people’s PA may alter their satisfaction with their sleep and warrant future research to test whether these changes in PA lead to changes in objectively assessed sleep characteristics, including sleep timing.

1.3.3.2 The Effect of NA on Sleep Characteristics

Five studies examined whether daytime NA predicted sleep continuity or duration on the corresponding night. Three studies showed no significant effects of NA (de Wild-Hartmann et al. 2013; Galambos et al., 2009; Scott & Judge 2006). In contrast, two studies showed significant effects of NA on sleep continuity, whereby on days individuals reported more NA relative to their average level, they subsequently experienced poorer sleep continuity (Brissette & Cohen, 2002; Kalmbach et al., 2014). One study showed significant effects of NA on sleep duration: Kalmbach et al. (2014) also reported that more daytime NA predicted shorter sleep duration. No studies have tested the effect of NA on sleep timing.

The results are thus mixed regarding the influence of NA on nighttime sleep characteristics. Preliminary findings show that greater NA levels on a given day may lead to poorer sleep continuity and, possibly, shorter sleep duration. These findings are consistent with evidence that the induction of mood changes prior to sleep, such as increased anxiety, can disturb sleep and lead to shorter sleep duration and poorer continuity (Tang & Harvey, 2004). Of note, while Kalmbach et al. (2014) reported that NA associated with both sleep continuity and duration, the authors did not test whether these effects were independent of one another. Half the studies found no significant effects of NA on sleep characteristics. Almost all of these studies were the same as those that tested the effects of PA on sleep characteristics and, as discussed earlier, the existing
literature is small, limited in assessment methods and statistical framework, and has not investigated effects of NA on sleep timing.

1.3.4 Summary

The extant literature suggests various links between sleep characteristics and affect, but the directionality of these associations and the specific characteristics of sleep that are related to affect have not yet been identified. Results regarding sleep duration are mixed, with half reporting null findings. These mixed results are inconsistent with previous experimental studies that show relatively consistent effects of sleep restriction and deprivation on mood. These findings may stem from several methodological limitations: studies relied on self-report measures of sleep and retrospective, once daily accounts of affect. In addition, most studies did not appropriately control for potential lag effects of sleep and mood, nor did they test for cumulative or nonlinear effects. Unlike the mixed results concerning sleep duration, there is consistent evidence that sleep continuity predicts changes in affect. However, existing studies are based on self-report sleep measures, which may be confounded by mood states, and it is unclear whether behaviorally quantified sleep continuity would similarly relate to affect. While there is evidence that individual differences in sleep timing associate with mood and that experimentally manipulated shifts in sleep timing lead to mood changes, studies have generally not tested whether there is a proximal, day-to-day relationship between sleep timing and affect. And, finally, relatively few studies have tested the effect of affect on sleep characteristics. Among those that have, only a few found significant associations. Overall, future studies should address the aforementioned limitations while considering the understudied effects of sleep timing on affect and the effects of affect on sleep.
1.4 Sleep Characteristics and Affect Reactivity

While studies have begun to reveal how individuals’ sleep characteristics and affect co-vary on a day-to-day basis, less is known regarding how changes in sleep characteristics may influence affective responses to daily experiences. While there are intrinsic processes, such as the circadian system, that regulate within-person variability in affect, there are also extrinsic, situational factors that can influence this variability. Such factors include everyday experiences that can be negative or positive, such as exposure to stressors or pleasant social interactions. Affect reactivity refers to changes in an individual’s levels of PA and NA that co-vary with the occurrence of these daily events (Mroczek & Almeida, 2004; Sliwinski et al., 2009; Stawski et al., 2008). Underlying these experience-contingent changes in affect are a series of neural and cognitive mechanisms involved in the processing of emotionally salient information and emotion regulation (Gross, 1998; Gross & Barrett, 2011). Studies on healthy adults show that experimentally-manipulated changes in sleep characteristics alter individuals’ reactivity to emotionally evocative stimuli (Franzen et al., 2008; Gujar et al., 2011; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). As an extension of this literature, emerging evidence suggests that people’s day-to-day variability in sleep characteristics associate with variability in their affect reactivity to naturally-occurring, daily experiences (Ong et al., 2013; Zohar, Tzischinsky, Epstein, & Lavie, 2005). The following sections will 1) further define affect reactivity and describe how an individual’s levels of PA and NA fluctuate in response to negative and positive daily experiences, 2) describe the influence of sleep characteristics on emotion processing and affect reactivity, and 3) review the extant literature on the effects of within-person variability in sleep characteristics on affect reactivity.
1.4.1 Affect Reactivity to Negative and Positive Daily Experiences

1.4.1.1 Negative Daily Events

A body of work has shown that everyday stressors can influence subjective wellbeing (Bolger, DeLongis, Kessler, & Schilling, 1989; Brissette & Cohen, 2002; Lee A Clark & Watson, 1988; Mroczek & Almeida, 2004; Ong et al., 2013; Sliwinski et al., 2009; Stawski et al., 2008; Watson, 1988; Zautra, Affleck, Tennen, Reich, & Davis, 2005). Studies have shown that on days participants reported more perceived stress, they also recalled experiencing a higher level of NA throughout that day (e.g., Stawski et al., 2008; Watson 1988). One approach to quantify affect reactivity is to assess whether day-to-day changes in an individual’s PA and NA covary with distinct events identified as stressors or negative events. Such stressors include experiences like high demands (e.g., workload, family obligations) and social conflicts (e.g., spousal argument, peer arguments). Greater changes in an individual’s levels of NA and PA following a stressor represent more NA reactivity and PA reactivity, respectively, to negative events.

Stress associates with increased NA, but effects on PA are less clear. Individuals tend to report a greater average level of NA on days they experience more frequent or more severe stressors relative to days with few or no stressors (Brissette & Cohen, 2002; Bolger et al., 1989; Mroczek et al., 2013; Sliwinski et al., 2009; Stawski 2008; Zohar et al., 2005). One study estimated that daily stressors together accounted for 20% of the variance in participants’ NA (Bolger et al., 1989). In contrast, several studies have found no association between exposure to daily stressors and PA (McIntyre et al., 1990; Stawski et al 2008; Zohar et al. 2005). Other studies have shown that daily stress predicts daily PA levels, albeit with effect sizes generally smaller than for NA (Clark & Watson 1988; Ong et al., 2013; Mroczek et al., 2013). For instance, Mroczek et al. (2013) reported that on days when participants experienced at least one social conflict, work stressor, or
home stressor, they reported relatively higher NA and lower PA in comparison to no-stressor days, with the effect on NA being greater ($\beta = 1.69$) than that on PA ($\beta = -0.18$). Overall, findings suggest individuals exhibit affect reactivity to daily stressors and more strongly with respect to NA than PA.

1.4.1.2 Positive Daily Events

Positive experiences can also alter an individuals’ affect, with evidence that when individuals experience positive social and work events they also experience increased in PA. Studies have found that when individuals engage in positive interpersonal events (e.g., played a game with others, parties, eating/drinking with others), they report greater PA (Clark & Watson 1988; McIntyre et al. 1990; McIntyre et al., 1991). In contrast, these same studies found participants’ NA levels unrelated to the occurrence of positive social experiences. Positive work experiences and events also influence PA. For instance, Zohar et al. (2003, 2005) collected daily information regarding participants’ PA, NA, and frequency of work events. Participants who reported more positive work events also reported higher PA relative to those with less positive work events. These patterns were consistent when examined on a within-person basis, such that individuals exhibited greater PA on days that they experienced more positive events (Zohar, Tzischinski, & Epstein, 2003; Zohar et al., 2005). Positive work activities were not correlated with NA in these studies. Collectively, these findings suggest that individuals exhibit affect reactivity, specifically in PA, to the occurrence of positive work and interpersonal events.
1.4.2 The Effects of Sleep Deprivation on Affect Reactivity

In order to test whether changes in individuals’ sleep characteristics can alter their affect reactivity, it is important to first understand mechanisms that underlie these affective responses. People undergo a series of unconscious and conscious cognitive processes that form their emotional response to a given stimulus or circumstance. Emotion regulation refers to these collective processes (Gross, 1998; Gross & Barrett, 2011). The process model of emotion regulation posits that individuals attend to emotionally salient information, cognitively appraise this information, and subsequently exhibit an emotional response (Gross & John, 2003; John & Gross, 2004). Any effects of sleep characteristics on affect reactivity would thus be reflected in either altered perceptions of an evocative stimulus or altered self-reported affect.

Several laboratory studies on healthy, young adults tested whether one-night of total sleep deprivation can change people’s affect reactivity ((Franzen et al., 2009; Franzen et al., 2008; Gujar et al., 2011; J. D. Minkel et al., 2012; Tempesta et al., 2010; Yoo et al., 2007). With the exception of one study that had participants engage in stressful tasks (Minkel et al., 2012), all studies used selected images from the International Affective Picture System (IAPS; Lang, 2005)) to serve as emotionally evocative stimuli. Images from the IAPS range in emotional valence with images being positive (e.g., happy people, animals, babies), negative (e.g., accidents, violent scenes, sick patients) or neutral (e.g., household objects, cars). Across studies, affect reactivity was assessed using various measures, including changes in pupil dilation, ratings of images, and self-reported PA and NA levels in response to the stimuli. Other studies that tested the effects of sleep stage restriction (e.g., restricted rapid eye movement sleep) or measured other forms of emotional responses (e.g., facial expressiveness to measure arousal) are beyond the scope of the current
review (Kahn-Greene, Lipizzi, Conrad, Kamimori, & Killgore, 2006; J. Minkel, Htaik, Banks, & Dinges, 2011; Rosales-Lagarde et al., 2012; Wagner, Fischer, & Born, 2002).

Studies showed a moderate-to-large effect of sleep deprivation on affect reactivity to negative stimuli (Franzen et al., 2008; Franzen et al., 2009). For instance, Franzen et al. (2008) found that sleep-deprived participants had greater pupil dilation when viewing negative images in comparison to control subjects. Because larger pupil dilation is thought to represent sustained emotional processing or reactivity (Bradley, Miccoli, Escrig, & Lang, 2008), these findings suggest greater affect reactivity to negative stimuli after sleep loss. In addition, sleep deprivation may amplify individuals’ affect reactivity to some stressful situations. Relative to a control group, participants who were sleep deprived reported more negative mood and subjective stress after engaging in a simple cognitive task designed to induce mild stress (Minkel et al., 2012). Interestingly, similar effects were not observed when subjects were administered a more difficult cognitive task, there was no significant difference between groups (Minkel et al., 2012). Regarding this latter result, perhaps sleep deprivation does not affect people’s reactions to situations that already elicit highly negative emotional responses. Taken together, evidence suggests that sleep deprivation can amplify people’s negative affect reactivity, at least in response to negative stimuli and milder forms of stress.

It is less clear whether sleep deprivation alters affect reactivity to positively-valenced images, with studies of significant result reporting small to moderate effect sizes. One study found sleep deprivation increased affect reactivity specifically to negative but not positive IAPS images (e.g., Franzen et al., 2008). In contrast, Gujar et al. (2011) found that when viewing images that ranged from neutral to extreme positive valence, sleep-deprived participants showed a moderate increase in the number of images they rated positive compared to their baseline ratings; control
participants did not exhibit this change. In addition, Tempesta et al. (2010) found participants rated positive images as more positive after sleep deprivation relative to baseline (Cohen’s $d = .40$). It is thus possible that sleep deprivation enhances reactivity to not only negative but also positive information, which suggests that sleep loss might enhance emotional lability.

Sleep loss may also influence how individuals perceive neutrally-valanced information (Tempesta et al., 2010; Yoo et al., 2007). For example, Tempesta et al. (2010 found that sleep-deprived participants reported more negative mood and also rated neutral images as more negative in comparison to baseline. The latter effect of sleep deprivation was moderate (Cohen’s $d = .58$), while control participants did not exhibit significant changes from baseline (Tempesta et al., 2010). In another study, participants were asked to rate images that ranged in valence from neutral to extremely negative. Sleep deprived participants rated a larger proportion of these images as negative in comparison to control participants (Yoo et al., 2007). Taken together, the evidence suggests that sleep deprivation can lead people to perceive neutral stimuli as if they were more emotionally valanced.

To date, experimental work has shown that complete sleep deprivation can influence people’s affect reactivity by changing how they perceive and respond to emotionally evocative information, mild stress, and neutrally-valenced information. The effect sizes of sleep deprivation on affect reactivity appear to vary in part by the emotional valence of stimuli (e.g., negative versus positive), with effects being relatively larger for negative stimuli. However, the varying effect sizes may also be due to differing measures of affect reactivity (e.g., pupil dilation versus image ratings). Of note, these studies were based on complete sleep deprivation and the magnitude of these effects may not generalize to naturally-occurring short sleep duration. Based on these findings, however,
it is predicted that on days after individuals sleep less than their average duration, they will exhibit relatively more affect reactivity to both positive and negative daily experiences.

1.4.3 The Day-to-Day Relationship between Sleep Characteristics and Affect Reactivity

While evidence from experimental studies suggests that sleep loss alters affect reactivity, less is known regarding how these relationships translate within the context of naturally-occurring sleep patterns and daily experiences. As previously reviewed, individuals generally experience more NA and possibly less PA on days they experience stressors (Bolger et al. 1989; Sliwinski et al., 2009; Stawski et al., 2008). In addition, individuals commonly report more PA following positive social interactions (Clark & Watson, 1988). If within-person, day-to-day variability in sleep duration impacts an individual’s affect reactivity, it is predicted that after an individual has shorter sleep duration, s/he will report more NA in response to daily life stressors and more PA in response to positive social interactions relative to days s/he sleeps longer.

While experimental studies have focused on sleep duration, it is possible that other sleep characteristics also influence affect reactivity. As reviewed previously, studies show that within-person variability in sleep continuity influences next day affect. Because daily fluctuations in affect occur in response to both positive and negative events, it is plausible that changes in an individual’s sleep continuity will also alter their affect reactivity to daily experiences. To date, two studies have investigated the relationship between sleep duration, sleep continuity, and affect reactivity outside of the laboratory setting.

Evidence from one study by Ong et al. (2013) shows that individuals with poorer sleep continuity also exhibit greater PA reactivity to negative and positive daily events. Participants were midlife adults who participated in a nationwide survey, and the study tested whether naturally-
occurring individual differences in sleep continuity associated with affect reactivity to daily experience. Affect reactivity and sleep measures were collected from two separate monitoring periods. Throughout an 8-day period, participants reported their daily PA levels and daily events via evening phone interviews. Sleep efficiency, a measure of sleep continuity, was derived from actigraphy data collected during a separate 7-day monitoring period that occurred, on average, a year apart from affect data collection. PA reactivity was quantified as the correlation between an individual’s self-reported levels of PA and number of negative (social conflicts, work stressors) or positive experiences (social interactions) on a given day. The authors tested the association between these correlations (derived slopes) and sleep continuity, while controlling for sleep duration. Although the authors included mean NA as a covariate in their study, they did not include measures of NA reactivity.

In this study, poorer sleep continuity was associated with greater PA reactivity. Overall, participants reported higher PA on days with more positive daily events (B =.05, SD =.04) and lower PA on days with more negative events (B =-.11, SD =.05). Poorer sleep continuity was associated with exaggerated reactivity (greater increase in PA with positive events, greater decrease with negative events). Taken together, the work by Ong et al. (2013) showed that poor sleep continuity is an important sleep characteristic, at least as an individual difference, that associates with greater PA reactivity to daily experiences outside the laboratory setting.

While findings from Ong et al. (2013) extend upon experimental work, the temporal parameters of this study and the specific measures used to quantify sleep continuity and PA reactivity bear on interpretation of findings. The difference between the sleep and affect monitoring periods precludes any interpretation that sleep continuity leads to changes in affect reactivity. In addition, while affect reactivity is conceptualized as changes in a person’s affect
following distinct daily events, this study quantified reactivity scores based on the covariation of a participant’s retrospective account of his/her daily PA level and overall daily experiences. Because participant’s PA and daily experiences were measured only once a day, the PA reactivity score does not indicate whether within-person changes in PA occur following specific events. Similarly, sleep continuity was measured as the individual’s average continuity across the monitoring period, which did not enable researchers to test whether day-to-day changes in sleep continuity led to changes in affect reactivity. Finally, because the authors did not report effects on NA reactivity, any effects of sleep continuity on NA reactivity are unknown. In sum, further work is warranted to test the within-person, temporal relationship between sleep continuity and affect reactivity, improve upon assessment of affect reactivity, and consider possible effects of sleep continuity on NA reactivity.

One approach to assess affect reactivity is to identify events throughout the day and determine whether individuals’ affect changes in conjunction with or in response to these events. To date, one field study has tested whether day-to-day changes in participants’ sleep characteristics predict their affect reactivity to daily experiences. In a sample of resident physicians, Zohar et al. (2005) assessed whether changes in the residents’ sleep characteristics resulting from an on-call (24-hr) shift predicted changes in their affect reactivity to work events. Research assistants called the residents 3x/day at random intervals to prompt them to complete questionnaires regarding their sleep the night before and their affect and work experience at the time of assessment. Positive events were defined as goal-enhancing experiences such as performing a novel professional task or managing a complex patient case. Negative events were defined as goal-disrupting events, including instances when another person disrupted the participants’ scheduled activity or when the residents experienced unforeseen difficulty in their scheduled activity. In other words, monitored
experiences were events that helped or hindered the participant from achieving their professional goals.

During the on-call shift, the residents worked overnight and slept in between tasks. As a result, they slept significantly less (4.3 ± 1 hr), as determined by actigraphy, during the on-call shift compared to the nights prior and after this shift (average: 7.2 ± 1.2 hrs). Residents’ actigraphy-derived sleep continuity was high across days, and was not significantly different between the on-call shift (93.3% ± 8.5% sleep efficiency) and the day prior (91.9% ± 8.4%) or the subsequent day (92.6% ± 8.3%). Another measure of sleep continuity--number of awakenings--was also assessed via self-report, and this measure of sleep continuity differed across nights, with residents reporting more awakenings when on-call. In terms of how their experiences related to their affect, positive (goal-enhancing) events were moderately associated with changes in PA but not NA, while negative (goal-disrupting) events moderately associated with NA but not PA.

The authors found that sleeping less and experiencing more self-reported awakenings had a small effect on NA reactivity. Specifically, when the residents experienced shorter sleep duration and more awakenings, they exhibited no change in NA in the absence of negative events (i.e. baseline) but more NA in the presence of negative events. In contrast, sleep characteristics were unrelated to PA in the presence of positive events. Interestingly, shorter sleep duration was minimally associated with less PA during the absence of positive work events (i.e., baseline). These findings suggest that sleep loss amplifies NA reactivity to negative events and elevates baseline PA levels but does not alter PA reactivity to positive events. Overall, this study extends experimental work in showing that day-to-day sleep characteristics can alter affect reactivity, at least in terms of NA, to work-related experiences.
There are methodological limitations in the study by Zohar et al. (2005) that should be considered in the interpretation of findings. First, participants were prompted at random intervals (3x/day) to report their affect levels and work experiences. Because the authors did not report whether they recorded and controlled for times of assessment, it is unclear if this study considered the potential influence of time of day. Because PA exhibits a diurnal rhythm (Murray et al., 2002), it is possible that this diurnal pattern in part underlies some of the observed fluctuations in affect. Second, participants were resident physicians and the observed sleep restriction and awakenings resulted from overnight, on-call shifts. It is possible that shifts in sleep time, rather than sleep loss or poor sleep continuity per se, led to the observed changes in affect reactivity. In addition, participants worked 6-10 nightshifts per month. Because shift workers experience circadian desynchrony (Monk, 2000), it is unclear whether the findings from this study are in part influenced by circadian disruptions that occurred from shifts in sleep time. Future studies are thus needed to account for the diurnal rhythm of affect and to test whether shifts in sleep time may contribute to reported effects on affect reactivity.

It is notable that the study by Zohar et al. (2005) was designed to monitor work-related experiences specific to resident physicians. By focusing on resident physicians, the researchers were able to test the effects of drastic changes in sleep characteristics outside the laboratory. In addition, researchers were able to compare participants’ affect in response to similar work demands before and after sleep changes. One limitation, however, is that monitored experiences were task-related events specific to this cohort and did not include measures of social interactions outside of task performance. Zohar et al. (2005) found that sleep characteristics enhanced participants’ NA reactivity to negative work events, but did not alter PA reactivity. Because individuals tend to exhibit more PA reactivity to positive social interactions in comparison to
stressors, it is possible that there were effects on PA reactivity not captured in the authors’ measure of experiences.

1.4.4 Summary

In summary, evidence suggests that within-person variability in sleep duration and sleep continuity can influence affect reactivity to daily experiences, while any effects of sleep timing are unknown. Experimental studies show that sleep loss can lead to exaggerated reactivity to both positive and negative stimuli and increase the likelihood that participants perceive neutral stimuli as emotionally salient. Only one naturalistic study has used repeated measures of affect and daily experiences to test how participants’ affect changes in relation to distinct events that occur throughout the day and whether the participants’ sleep characteristics modify this reactivity. Findings from this study by Zohar et al. (2005) demonstrate that changes in sleep duration and sleep continuity not only predict an individual’s overall affect but can also predict PA reactivity to daily events. However, because this study did not control for time of day, it is possible that these results may be partly confounded by the diurnal rhythm of PA. In addition, it remains unclear whether sleep characteristics modify how individuals’ PA levels change in response to a wider range of daily experiences, including social interactions. No studies to date have tested whether changes in sleep characteristics modify how an individual’s NA levels change in response to daily events. Last, it is unknown whether shifts in sleep timing, such as those experienced by the participants in the study by Zohar et al. (2005) also associate with changes in affect reactivity.
1.5 Literature Summary and Future Directions

The extant literature begins to support a bidirectional relationship between sleep characteristics and mood. However, results are either mixed or unclear regarding how specific sleep characteristics associate with affect and the directionality of these associations. There is relatively consistent evidence to suggest that changes in sleep continuity lead to changes in affect, at least in terms of perceived sleep continuity, whereas results regarding sleep duration are mixed. There is a paucity of work on sleep timing, with only one study incorporating this sleep characteristic. In terms of a potential bidirectional relationship, relatively fewer studies have tested the prospective effects of affect on sleep characteristics and only a few of these studies report statistically-significant effects.

There is growing evidence to suggest that changes in sleep characteristics can lead to changes in affect reactivity. While experimental evidence suggests that sleep deprivation amplifies people’s affective responses to emotionally evocative stimuli, few studies have tested whether these findings generalize outside the laboratory. Thus far, one study has shown that poorer sleep continuity and less sleep duration predict more PA reactivity to work experiences. However, it is unknown if sleep timing similarly associates with PA reactivity, whether these findings extend to affect reactivity to social experiences aside from work-specific events, and whether any sleep characteristics predict NA reactivity.

Future studies can test the bidirectional association between sleep and affect, and the effects of sleep characteristics on affect reactivity by addressing methodological limitations that overlap both sets of literature. Specifically, studies can incorporate: 1) sleep timing, an understudied sleep characteristic relative to affect; 2) actigraphic measures of sleep, for objective assessment and to avoid the potential confound of mood states with recall bias; 3) repeated,
momentary measures of affect to avoid recall bias, appropriately control for the diurnal rhythm of affect, and with respect to affect reactivity, assess affect proximal to experienced events; 4) measures of daily social interactions in addition to work experiences, and 5) a statistical framework that appropriately controls for potential lag effects of sleep and affect, and tests the cumulative and nonlinear effects of sleep where plausible.

1.6 Current Study Aims

The current study aims were two-fold. The first aim was to test for bidirectional relationships between sleep and affect; that is, whether: a) sleep characteristics (duration, continuity, timing) on a given night predict next day levels of PA and NA; and b) daytime affect predicts sleep characteristics on the corresponding night. The second aim was to test whether sleep characteristics on a given night predict next day affect reactivity to daily experiences.

In regard to Aim 1, this study incorporated a range of methodologies and statistical analyses designed to extend upon previous literature. To the best of our knowledge, this was the first study to test the effects of naturally-occurring shifts in sleep timing on both PA and NA, and vice versa. This study was also the first to use a combination of ecological momentary assessment (EMA) and actigraphy methods to assess affect and sleep characteristics, respectively. In addition, the current study used a hierarchical linear modeling (HLM) framework that includes lag effects of both sleep characteristics and affect in its models and will include secondary analyses to test for cumulative and nonlinear effects of sleep characteristics on affect.

In regard to Aim 2, the current study incorporated similar improvements in design, methodology, and statistical analyses to extend upon previous literature. To the best of our
knowledge, this was the first study to test the association between sleep timing and affect reactivity in a naturalistic setting. This study was the first to test the effects of within-person changes in sleep characteristics on NA reactivity, and examined affect reactivity to a wider range of social experiences (positive and negative social interactions, work strain) than in previous research. While previous research has incorporated repeated measures of affect and daily experiences, this study used EMA to more comprehensively collect these data at fixed intervals set from each participant’s wake time until bedtime. This protocol provided more thorough assessment of participant experiences and changes in affect as they occur in daily life and, by recording the time of assessment relative to participants’ awakening, allowed us to control for the influence of diurnal rhythms in affect. Specific aims and hypotheses are outlined below:

Specific Aim 1a) Determine whether sleep characteristics (duration, continuity, timing) on a given night predict next-day PA and NA. Based on previous literature, we hypothesized that:

1. After individuals sleep less (shorter duration) and have poorer sleep continuity than their average, they will report more NA and less PA the following day.
2. Sleep timing will associate with next-day affect nonlinearly, such that when individuals deviate in their sleep timing (either earlier or later than their average) they will then report more NA and less PA the following day.
3. There will be a cumulative effect of sleep duration over time on affect. When individuals sleep less than their usual sleep duration for two consecutive nights or more, they will then exhibit greater affect changes (more NA, less PA) in comparison to a single night of short sleep duration.
Specific Aim 1b) Test whether daytime levels of affect (PA and NA) predict nighttime sleep characteristics (duration, continuity, timing). Based on previous literature, we hypothesized that:

1. Greater average NA and lower average PA will predict shorter sleep duration, poorer sleep continuity, and later sleep timing that night.

Specific Aim 2) Test whether sleep characteristics (duration, continuity, timing) on a given night predict next-day affect reactivity to positive and negative daily experiences. Based on previous experimental and cohort (medical resident) studies, we hypothesized that:

1. Sleep duration, continuity, and timing will interact with daily positive and negative experiences. After nights of shorter sleep duration, poorer sleep efficiency, and shifts in sleep timing (either earlier or later), individuals will exhibit:

   a. Greater increases in NA following negative experiences (e.g., higher NA following high work strain, negative social interactions).

   b. Greater decreases in PA following negative experiences (e.g., lower PA following high work strain, negative social interactions), albeit these effects will be smaller than predicted under 1a.

   c. Greater change in PA following positive experiences (e.g., greater increase in PA following positive social interactions).
2.0 Methods

2.1 Participants

The current study will draw from previously collected data. Participants were 490 midlife men and women from the Adult Health and Behavior Project phase 2 (AHAB-II), a study of psychological, behavioral, and biological risk factors for subclinical cardiovascular disease in healthy individuals. Participants were recruited between March 2008 and October 2011 through mass mailings of recruitment letters to individuals randomly selected from voter registration and other public domain lists. Participant informed consent was obtained in accordance with the guidelines of the University of Pittsburgh Institutional Review Board (IRB).

To be eligible to participate in AHAB-II, individuals had to be between the ages of 30 and 54 years and working at least 25 hours per week outside the home (this latter restriction due to a substudy focusing on occupational stress). Individuals were excluded from participation if they a) had a history of clinically apparent cardiovascular disease, schizophrenia or bipolar disorder, chronic hepatitis, renal failure, neurological disorder, lung disease requiring drug treatment, or Stage 2 hypertension (systolic/ diastolic blood pressure ≥160/100 mm Hg); b) excessively consumed alcohol (≥5 portions, 3-4 times per week); c) used fish oil supplements (because of the requirements for another substudy); d) were prescribed use of insulin, glucocorticoid, antiarrhythmic, antihypertensive, lipid-lowering, psychotropics, or prescription weight loss medications; e) were pregnant; or f) were shift workers. Participants signed an IRB-approved informed consent agreement when enrolled and received compensation up to US$410, depending on extent of participation in study visits and protocol compliance.
2.2 Procedure

As part of the larger AHAB II study, participants completed seven laboratory visits designed to gather a wide range of information including psychosocial, behavioral, biological, neuropsychological, and neuroimaging data. Participant demographics and baseline depression scores were collected over the course of these visits and will be used as covariates in the current study (see below).

Participants completed a field (i.e., non-laboratory, home and work environment) monitoring session between Visits 2 and 3. During this time, data regarding subjects’ daily sleep, affect, and psychosocial experiences were collected. Actigraphy data were collected for 7 days (see below) to assess sleep. Four of these days were ecological momentary assessment (EMA) monitoring days, and included three workdays and one non-workday. On monitoring days, participants were instructed to indicate when they awoke using a personal digital assistant (PDA; Palm Z22, software: Satellite Forms). The PDA then prompted participants at hourly intervals, set from time of awakening, to complete a 43-item questionnaire. This questionnaire contained affect and daily experience items described below. Participants received extensive training and practice using the PDA and received feedback on compliance following a practice day. Additionally, participants were phoned four times throughout their time in the field for technical support. During this monitoring period, other data including saliva samples for measurement of cortisol and ambulatory blood pressure were collected, but are not relevant to the current study.
2.3 Measures

2.3.1 Positive and Negative Affect

Participants were administered an adapted version of the Positive Affect Negative Affect Schedule-Short Form (PANAS-SF; Thompson, 2007)) on an hourly basis. In this version, participants rated 13 affect items on a six-point scale. The items “ashamed,” “active” and “alert” were deleted a priori from the scale due to rotated principal components analysis performed on previous samples, which revealed low factor loadings on these items. Additional items, “happy” and “cheerful” from the Profile of Mood States scale (POMS; McNair et al., 1981) were added to represent PA terms with low arousal associations. The resulting survey included “inspired,” “determined,” “attentive,” “happy,” and “cheerful” items in the PA scale. In addition to four NA items (“upset,” “hostile,” “nervous,” “afraid”) from PANAS-SF, three items (“angry,” “lonely”, “sad”) were added a priori from the PANAS-X in order to include items that measured sadness and anger as well as anxiety. For the purposes of this study, two measures of PA and NA will be derived from these data: 1) a measure of daily PA and NA, calculated as the averages of PA and NA ratings endorsed throughout a given day. These daily levels of PA and NA will be used in Aims 1 and 2. And 2), the momentary ratings of PA and NA will be used for Aim 3 to assess the association between daytime experiences and affect rating (i.e., affect reactivity) at each hourly interval.
2.3.2 Daily Events

In addition to affect items, participants completed an hourly report regarding their work and social experiences at the time of or preceding the momentary assessment. Items will be derived from various scales included in the hourly assessments to form measures of work Demand and Control, as well as Positive and Negative social interactions.

Measures of momentary Demand and Control will be derived from two scales. The three-item Task Demand scale (i.e., “Required working hard?” “Required working fast?” and “Juggling several tasks at once?”) and the two-item Decisional Control scale (i.e., “Could change activity if you chose to?” and “Choice in scheduling this activity?”) are based upon comparable scales from the Job Content Questionnaire (JCQ; Karasek, 1985). For the purposes of momentary assessment, these items were revised to reflect activities “during the past 10 minutes” both in and out of the workplace. Participants responded to each of these items using a 6-point Likert scale (NO! No no yes Yes YES!). We created a dichotomized measure of task strain by assigning a score of “1” to observations above the sample median in Demand and less than or equal to the sample median in Control. A score of “0” will be assigned to all other periods. This approach allowed us to identify events characterized as high in demand and low in latitude, and test whether an individual’s sleep characteristics modify how his/her affect changes in response to these moments of stress.

Through EMA, participants were also administered several items regarding their most recent social interaction. Participants indicated the time of interaction, number of people involved, and types of partners involved (e.g., spouse, coworker). To reduce the possibility of redundancy, we will eliminate scores for any interaction occurring more than 45 minutes before each hourly interview. For the purposes of the current study, scores involving any partner will be considered (i.e., no distinction between spousal or work partners). Social interaction quality was assessed
through 4 Likert-scale items: two assessed positive aspects of interactions (e.g., “agreeable interaction”, “pleasant”) and two assessed negative aspects of interactions (e.g., “someone in conflict with you”, “someone treated you badly”). Item responses [NO! No no yes Yes YES!] were converted to a 1-6 rating scale. Here, we derived two continuous scores: a pleasant interaction score was calculated as the average score of the two positive items, and a social conflict score was derived as the average of the two negative items.

2.3.3 Sleep Characteristics

Participants were asked to wear Actiwatch-16 (Bend, OR: Philips Electronics), a wrist accelerometer, which samples movement several times per second. Throughout a 7-day period that overlapped with the 4-day EMA monitoring period, participants wore the Actiwatch 24-hours a day and were instructed to keep the watch on even when showering. The monitoring period included at least one night preceding a free (i.e., non-work) day to capture differences between sleep intervals preceding work and free days. Data were saved in 1-minute epochs and scored with Actiware software (v5.59; Murrysville, PA) using automated, standard medium thresholds: Sleep onset was defined as a period lasting at least 10 consecutive minutes with <40 counts of activity (i.e., movement) per epoch. Wake onset was defined as 10 consecutive minutes of ≥40 activity counts per epoch.

In addition to using automated thresholds to determine sleep intervals, we had two research assistants and one graduate student (P.W.) examine each participant’ actigraphy data to detect instances where the program may have erroneously detected or omitted a sleep interval. For instance, there were cases in which participants stopped wearing their actiwatch because they completed the study protocol but did not return to the lab for a day (or more). As a result, the
actiwatch remained activated and the software detected an extended time of inactivity. In such cases, the software was unable to accurately determine the beginning and/or end point for a given day and a given sleep interval. To correct such errors, we visually inspected the data to infer when the participant last took off the watch in accordance with laboratory records of when participants completed the protocol and returned the actiwatch. We manually excluded these interval(s) in which the actiwatch was active but the participant was done with the monitoring period. After inserting such exclusions, we reran the automated scoring and the software program was subsequently able to detect and score the sleep interval.

Actiwatch data were used to quantify three sleep characteristics of interest. Sleep duration was defined as the total time between sleep onset and wake onset. As an estimate of sleep continuity, sleep efficiency was calculated as the percentage of the total rest interval scored as total sleep time, minus non-sleep time. Finally, the midpoint of sleep was calculated as an estimate of sleep timing, and was defined as the midpoint between sleep onset and wake onset.

The current study involves two forms of these actigraphy data. First, to consider individual differences in sleep characteristics, we calculated each participant’s average sleep characteristics, based on averaged values across all available data in the monitoring period (including those outside the EMA monitoring period). We included these average values as covariates. Second, we examined the participant’s sleep characteristics on the night preceding and following each EMA monitoring day to test the daily relationships between sleep and affect.
2.3.4 Covariates

2.3.4.1 Demographics

The participants reported their age, sex, and race/ethnicity. These variables will be used as covariates in all analytical models.

2.3.4.2 Depressive Symptomatology

In order to adjust for possible individual differences in baseline mood, depressive symptomatology was measured using the Center for Epidemiological Studies-Depression (CESD) scale (Radloff, 1977) and will be included as a covariate in the current study. This 20-item measure assesses how frequently subjects experienced a range of psychological and physical symptoms of depression during the past week. Responses are on a 4-point scale ranging from 0 (rarely or none of the time [<1 day]) to 3 (most or all of the time [5 to 7 days]). Higher scores indicate more severe depressive symptomatology, with a maximum score of 60. The CESD has high internal consistency (Cronbach α = 0.87; Radloff, 1977). To avoid confounding sleep problems and depression symptoms, the total score minus the sleep item will be used.

2.4 Statistical Analyses

Statistical analyses were performed with IBM SPSS Statistics (version 26). Prior to testing, study variables will be examined for outliers and to verify assumptions of normality. Generally, outliers (> +/- 3 SD from the mean) will be removed. Natural log transformations will be conducted on data deviating from the acceptable range of skewness and kurtosis (+/- 2.0).
The data collected involved repeated measures of sleep, affect, and psychosocial experiences. To account for this nested data framework, hierarchical linear models (HLM) will be used to conduct all primary analyses. HLM allows for the study of within-person changes over time while considering possible differences between persons that may confound the outcomes of interest (Bolger & Laurenceau, 2013). Three sets of analyses were completed to address each study aim. As shown in Table 3, each model involved either two or three nested layers of data. All models included age, sex, race, baseline depressive symptomatology, and average sleep characteristics (duration, continuity, timing) as covariates. Details regarding each set of analyses are outlined below according to Specific Aim:

1. **We tested whether sleep characteristics (duration, continuity, and timing) on a given night predict affect (PA and NA) the following day.** As shown below, the models have two levels, and lag effects of previous day PA and NA are included. Each sleep characteristic term in Level 1 is person mean-centered in order to test the effect of within-person changes, while participants’ average sleep characteristics are included in Level 2 to control for potential effects of individual differences. As shown below, models simultaneously test for the effects of all three sleep characteristics. If, however, there are issues of multicollinearity, separate models will be tested as needed. The following equations test linear effects of sleep characteristics on affect. We also tested secondary models that include quadratic terms for a given sleep characteristic in Level 1 to test for nonlinear effects. In addition, we aimed to conduct exploratory analyses to test whether there may be a cumulative effect of sleep duration on affect.

   **Level 1**

   \[
   PA_{t1} = \beta_0j + \beta_{1j}(\text{Duration-Avg Duration})_{t-1} + \beta_{2j}(\text{Continuity-AvgContinuity})_{t-1} + \\
   \beta_{3j}(\text{Timing-AvgTiming})_{t-1} + \beta_{4j}PA_{t-1} + \varepsilon_{ij}
   \]
NA_{t1} = \beta_{0j} + \beta_{1j}(\text{Duration - Avg Duration})_{t-1} + \beta_{2j}(\text{Continuity - Avg Continuity})_{t-1} + \beta_{3j}(\text{Timing - Avg Timing})_{t-1} + \beta_{4j}N_{t-1} + \epsilon_{ij}

Level 2

\beta_{0j} = \gamma_{00} + \gamma_{01}\text{Age} + \gamma_{02}\text{Sex} + \gamma_{03}\text{Race} + \gamma_{04}\text{AvgDuration} + \gamma_{05}\text{AvgContinuity} + \gamma_{06}\text{AvgTiming} + \mu_{0j}

\beta_{1j} = \gamma_{10} + \gamma_{11}\text{Age} + \gamma_{12}\text{Sex} + \gamma_{13}\text{Race} + \gamma_{14}\text{AvgDuration} + \gamma_{15}\text{AvgContinuity} + \gamma_{16}\text{AvgTiming} + \mu_{1j}

\beta_{2j} = \gamma_{20} + \gamma_{21}\text{Age} + \gamma_{22}\text{Sex} + \gamma_{23}\text{Race} + \gamma_{24}\text{AvgDuration} + \gamma_{25}\text{AvgContinuity} + \gamma_{26}\text{AvgTiming} + \mu_{1j}

2. We tested whether affect (PA and NA levels) on a given day predict sleep characteristics (duration, continuity, and timing) that corresponding night. As shown below, the models have 2-levels and lag effects of previous night sleep characteristic are included in the model. Each affect term in Level 1 is person mean-centered in order to test the effect of within-person changes, while participants’ average affect values are included in Level 2 to control for potential effects of individual differences.

Level 1

Duration_{t1} = \beta_{0j} + \beta_{1j}(\text{PA - AvgPA})_{t1} + \beta_{2j}(\text{NA - AvgNA})_{t1} + \beta_{3j}\text{Duration}_{t-1} + \epsilon_{ij}

Continuity_{t1} = \beta_{0j} + \beta_{1j}(\text{PA - AvgPA})_{t1} + \beta_{2j}(\text{NA - AvgNA})_{t1} + \beta_{3j}\text{Continuity}_{t-1} + \epsilon_{i}

Timing_{t1} = \beta_{0j} + \beta_{1j}(\text{PA - AvgPA})_{t1} + \beta_{2j}(\text{NA - AvgNA})_{t1} + \beta_{3j}\text{Timing}_{t-1} + \epsilon_{i}

Level 2

\beta_{0j} = \gamma_{00} + \gamma_{01}\text{Age} + \gamma_{02}\text{Sex} + \gamma_{03}\text{Race} + \gamma_{04}\text{AvgPA} + \gamma_{05}\text{AvgNA} + \mu_{0j}

\beta_{1j} = \gamma_{10} + \gamma_{11}\text{Age} + \gamma_{12}\text{Sex} + \gamma_{13}\text{Race} + \gamma_{14}\text{AvgPA} + \gamma_{15}\text{AvgNA} + \mu_{1j}

\beta_{2j} = \gamma_{20} + \gamma_{21}\text{Age} + \gamma_{22}\text{Sex} + \gamma_{23}\text{Race} + \gamma_{24}\text{AvgPA} + \gamma_{25}\text{AvgNA} + \mu_{1j}
3. We tested whether sleep characteristics (duration, continuity, timing) on a given night predict affect reactivity the next day. Specifically, we tested whether each of the three sleep characteristics moderate the effects of daily experiences (task demand, negative social interaction, positive social interaction) on affect (PA and NA). These models have three levels (Level 1= Moment-to-Moment, within-day, within-person; 2 = Day-to-Day, within-person, 3 =Between Subjects). Below is an example of one model testing the effect of sleep duration on PA reactivity to social conflicts; Sleep =Sleep Duration, Conflict =Negative Social Interaction. In Level 1, Conflict is person mean-centered in order to test for the effect of changes in conflict level on PA. A time-centered variable is also included in Level 1 in order to control for time-of-day effects on affect. In Level 2, both main effects of sleep duration and conflict, and an interaction term of sleep*conflict are included to test whether there sleep duration modifies the effect of conflict on PA variability.

**Level 1**

\[ PA_{ij} = \Pi_{0ij} + \Pi_{1ij}(Conflict-Avg \ Conflicts) + \Pi_{2j}(Time-Midday)_{ij} + \varepsilon_{ij} \]

**Level 2**

\[ \Pi_{0ij} = \beta_{00j} + \beta_{01j}Sleep_{i-1} + \beta_{02j}(AvgConflict)_{ij} + \beta_{03j}(Sleep_{ij-1}*AvgConflict) + \beta_{04j}Day + r_{0ij} \]
\[ \Pi_{1ij} = \beta_{10j} + \beta_{11j}Sleep + r_{1ij} \]
\[ \Pi_{2ij} = \beta_{20} + r_{2ij} \]

**Level 3**

\[ \beta_{00j} = \gamma_{00} + \gamma_{01}Age + \gamma_{02}Sex + \gamma_{03}Race + \gamma_{04}AvgSleep + \mu_{0j} \]
\[ \beta_{01j} = \gamma_{10} + \gamma_{11}Age + \gamma_{12}Sex + \gamma_{13}Race + \gamma_{14}AvgSleep + \mu_{1j} \]
\[ \beta_{10j} = \gamma_{100} + \gamma_{101}Age + \gamma_{102}Sex + \gamma_{103}Race + \gamma_{104}AvgSleep + \mu_{1j} \]

(Similar equations for each of the other \( \beta \)-values not shown)
2.4.1 Exploratory Analyses

When there were significant individual differences in the effects of sleep characteristics on affect, or vice versa, we tested whether various participant characteristics might contribute to these differences. For instance, if there were significant individual differences between participants in how sleep duration affects NA, we would test whether participants’ chronotype moderates the effects of sleep duration on NA. Specifically, we examined seven characteristics that have previously been associated with mood and sleep characteristics, including age, sex, race, years of school and family income as indicators of socioeconomic status, neuroticism, and chronotype (Duggan, Friedman, McDevitt, & Mednick, 2014; Dunlop, Song, Lyons, Manheim, & Chang, 2003; M. H. Hall et al., 2009; Hidalgo et al., 2009; Hume, Van, & Watson, 1998; Jorm, 2000; Nolen-Hoeksema, 2001; Roenneberg, Wirz-Justice, & Merrow, 2003; Schmitz, Kugler, & Rollnik, 2003; Stamatakis, Kaplan, & Roberts, 2007; Yoon et al., 2003). Neuroticism was assessed via the Revised NEO Personality Inventory (NEO-PI-R; Costa Jr & McCrae, 2008), and chronotype was assessed via the Composite Morningness Scale (CSM; Smith, Reilly, & Midkiff, 1989). The remaining demographic characteristics were each assessed through self-report items.
3.0 Results

3.1 Participant Characteristics

Of the 490 participants, 22 participants were missing actigraphy data. Among these 22 participants, four also had missing electronic diary data. In addition, six participants were missing baseline depression scores (CES-D scale). A total of 462 participants were included in all primary analyses. Among these participants, there were 1724 total observations in which a participant had both affect data for a given day and sleep data the preceding night (for Analyses 1 and 3). Regarding Analysis 2, there were 1691 total observations in which a participant had both sleep data for a given night and affect data for that corresponding day.

Less than 1% of the observations (17 for Analyses 1 and 3, 7 for Analysis 2) were excluded from analyses because these observations involved sleep midpoint data that were extreme outliers (>3SD from the average of the total sample) and skewed the data. For example, on two nights (one work, one non-work), a participant slept on average at 12:34AM and woke at 7:56AM. This led to an average midpoint of 4:15AM on these nights, which fell within the normal sample distribution. One two other workdays, however, the participant slept on average at 7:58PM and woke at 3:17AM, which led to a midpoint of 11:37PM. The participant’s sleep midpoint on the latter two nights were >3SD earlier than the sample mean. To account for potential effects of these statistical outliers, we excluded these observations from primary analyses. After these exclusions, a total of 1707 observations were included for Analysis 1 and 3, and 1684 for Analysis 2.

Table 4 lists participant characteristics and bivariate correlations of each characteristic with sleep duration, sleep midpoint, and sleep efficiency. All values represent averages across all
participants, with correlations referring to between-person correlations. While participants completed 4 days (3 work, 1 non-work) of EMA monitoring, participants tended to complete more days of actigraphy monitoring. On average, we obtained 7 nights of actigraphy data (range: 1-11 nights). Specifically, 51% of participants completed 7 nights, 20.3%, 1-6 nights and 39%, 8-11 nights. Average sleep characteristics presented were calculated as the average across all available data. As shown, several demographic characteristics such as sex, education, and marital status were related to one or more of these sleep characteristics (p’s >.05). Shorter sleep duration was correlated with later sleep midpoint (r =-.09, p <.05) and poorer sleep efficiency (r =.22, p <.01). However, sleep efficiency and midpoint were unrelated (p >.05). Average sleep midpoint and efficiency were both associated with participants’ smoking status and average physical activity levels (p’s <.05). Average sleep characteristics were unrelated to baseline depression and levels of positive and negative affect (p’s >.05).

3.2 Aim 1: The Effect of Sleep Characteristics on Affect

In our sample, participants tended to rate high on PA, low on NA, and showed moderate variability in affect as assessed on hourly intervals throughout the day: Participants reported an average PA of 4.0 (top tertile of sample >4.3, bottom tertile < 3.6; SD =0.7;), and an average NA of 1.9 (top tertile > 2.2; bottom tertile < 1.3; SD =0.7). We calculated the intra-class correlations (ICC) of affect variables to estimate within-person variability in the outcomes. A substantial proportion of affect variability was attributed to day-to-day variation among participants, with 77.7% of the total variance in PA and 87.3% in NA due to within-person variability. Next, we conducted baseline models to estimate the fixed effects of covariates (demographic, baseline
depression, and health behaviors) on affect. As shown in Tables 5 and 6, greater baseline depression was associated with less PA (B =-.03, p <.001) and more NA (B =.03, p <.001), and non-work days were associated with greater PA (B =.09, p<.001) and less NA (B =-.06, p <.001) relative to workdays. Other health behaviors and demographic characteristics were unrelated to PA and NA (p’s >.05).

3.2.1 The Effects of Sleep Characteristics on Affect

Day-to-day variation in sleep midpoint, sleep duration, and sleep timing did not significantly predict PA (p’s>.05; see Tables 7,9,11). The effects of day-to-day variation in sleep duration on NA trended towards significance (B =.01, p =.047; Table 10). When participants slept one hour longer on a given night relative to their average sleep duration, they tended to report a .01 increase in NA score. There were no significant effects of sleep midpoint or timing on NA (Tables 8, 12).

3.2.2 Individual Differences in the Effects of Sleep Characteristics on Affect

There were significant individual differences in the effects of sleep midpoint on affect. Some participants had weaker and some stronger negative associations between sleep midpoint and PA (B range: -.04–0.0, Wald Z =2.18, p =0.029; Table 7). Some participants had weaker and some stronger positive associations between sleep midpoint and NA (B range: 0.0–.04, Wald Z =3.33, p =0.001; Table 8). While there were statistically significant individual differences in the effects of sleep duration on PA and NA, and of sleep efficiency on NA, the variance estimates were negligible (estimates =.00, Wald Z =2.03- 2.48, p =.013-.043; Tables 9, 10, 12), and thus
cannot be interpreted. There were no individual differences in the sleep efficiency-PA association (p > .05; Table 11).

### 3.2.3 Exploratory Analyses

As noted, baseline depression was significantly associated with both PA and NA and may thus partly account for variance in affect explained by sleep characteristics. Thus, we ran an exploratory set of analyses (results not shown) omitting baseline depression in the model but found that all results persisted. We also conducted additional analyses to test for possible interaction effects between sleep characteristics on affect (results not shown). This allowed us to test, for instance, whether a combination of greater shifts in sleep midpoint and shorter sleep duration predicts greater decreases in NA. However, there were no significant interacting effects between any of the sleep characteristics on either PA or NA (p’s > .05). In order to consider nonlinear effects of sleep characteristics on affect, we conducted additional analyses that included quadratic terms of each respective sleep characteristic (results not shown). Findings showed no significant quadratic relationship between any of the sleep characteristics and affect (p’s > .05).

### 3.2.4 Unexplored Analyses

We initially aimed to test for the cumulative effects of sleep duration on affect. However, while all participants completed 4 days of EMA, they did not complete the EMA protocol on consecutive days. We were thus unable to create cumulative sleep scores and to explore possible cumulative effects.
3.2.5 Summary

Overall, we found that while participants exhibited a relatively large proportion of day-to-day variability in PA and NA, sleep characteristics on a given night did not significantly account for next day levels of affect.

3.3 Aim 2: The Effects of PA and NA on Sleep Characteristics

We calculated the intra-class correlations (ICC) of sleep characteristics to estimate within-person variability in the outcomes. Participants showed moderate levels of night-to-night fluctuation in sleep midpoint and efficiency, with 49.7% of the total variance in sleep midpoint and 40.8% in sleep efficiency attributed to within-person variability. Participants showed relatively less night-to-night fluctuations in sleep duration, with 16.9% of the total variation due to within-person variability.

We next estimated the fixed effects of covariates on sleep characteristics. Older age was associated with earlier sleep midpoint (B=-.02, p<.001), while current smokers tended to have later sleep midpoints compared to non-smokers (B =.35, p =.001; Table 13). Women (B =.37, 1.38, p’s <.05) and white participants (B = -.28, -1.60, p’s <.05) had longer sleep duration and greater sleep efficiency in comparison to men and non-whites, respectively (Table 14 & 15). Less physical activity was associated with greater sleep efficiency (B =-.00, p=.002). All sleep characteristics were positively correlated with corresponding sleep characteristics the preceding night (B’s: .12-.43, p’s<.05).
3.3.1 The Effects of PA and NA on Sleep Characteristics

Day-to-day shifts in PA were associated with sleep midpoint such that an increase in PA on a given day predicted a later sleep midpoint that corresponding night (Table 16, B = .23, p = .012). However, there were no significant effects of PA on sleep duration or sleep efficiency (p’s > .05; Tables 18 & 20). Regarding NA, day-to-day shifts in NA did not significantly influence any of the sleep characteristics (p’s > .05; Tables 17, 19, 21).

3.3.2 Individual Differences in the Effects of PA and NA on Sleep Characteristics

There were some individual differences in regard to the effects of PA. Some participants had a positive association, and others a negative association, between PA and sleep midpoint (B range: -1.06 - 1.53, Wald Z = 2.31, p = .021; Table 16). Similarly, participants also significantly differed in how their daytime PA predicted their sleep duration (B range: -1.81 - 1.59, Wald Z = 2.11, p = .035; Table 18). In regard to the effects of PA on sleep efficiency, our original model tested for individual differences in this relationship, but inclusion of a random effects term resulted in a Hessian error and the model did not run. Our final model thus did not test for such individual differences. Finally, participants did not show significant individual differences in the effect of NA on any of the sleep characteristics (p’s > .05; Tables 17, 19, 21).
3.3.3 Summary

Overall, we found that participants exhibited a small to large amount of day-to-day variability across sleep duration, continuity, and timing. We found that PA predicted later sleep timing, but there were no other significant effects of PA or NA on the sleep characteristics.

3.4 Aim 3: Sleep Characteristics and Affect Reactivity

Across participants, there were 22841 cases (1-hour bins) available in which participants had complete data regarding work demand/latitude and affect ratings, as well as corresponding sleep data from the prior night. The total sample median for work demand was 3.00 (mean: 2.95, SD: 1.22, range: 1.00-6.00). Demand scores between 3.10-6.00 were coded as high demand (1), and scores 3.00 and below were coded as low demand (0). The total sample median for work latitude/control was 4.00 (mean: 4.19, SD: 1.27, range: 1.00-6.00). Latitude scores 4.00 and below were coded as low latitude (1), and scores 4.10-6.00 were coded as high latitude (0). Overall, high demand and low latitude were modestly correlated ($r = .23$, $p < .001$).

Across participants, there were 15310 cases (1-hour bins) available in which participants indicated they had at least one social interaction, affect ratings, and corresponding sleep data from the previous night. The included social interaction cases were those that occurred immediately (up to 10 minutes) before the EMA assessment.

We conducted baseline models to estimate the fixed effects of covariates (demographics, baseline depression, health behaviors) on hourly measures of affect. In addition to average alcohol and smoking habits, we also included hourly measures of alcohol, caffeine, drug, and cigarette use.
to control for the proximal effects of substance use on affect. As shown in Tables 19 and 20, greater baseline depression was associated with lower levels of PA (B = -.03, p < .001) and higher levels of NA (B = .03, p < .001). Non-workdays were associated with higher PA (B = .08, p < .001) and lower NA levels (B = -.05, p < .001) relative to workdays. While individual differences in average alcohol use were not related to hourly PA or NA (p’s > .05), use of alcohol during a given hour-bin predicted both higher PA and lower NA levels during the same hour (B = .29, p < .001; B = -.10, p < .001, respectively). Drug use within the same hour was related to lower PA (B = -.11, p = .014). Finally, time of assessment was related to both PA and NA such that there was a significant quadratic relationship between the time of day and both affects (p’s < .001).

3.4.1 The Effects of Sleep Characteristics and Work Demand on Affect

3.4.1.1 Work Demand, PA and NA

Across analyses, work demand was not associated with PA (p’s > .05), but there were individual differences such that some participants exhibited a stronger relationship between demand and PA than others (estimates of variance = .05, p’s < .001; Tables 24, 26, 28). High work demand during a given hour was related to greater NA during the same hour (B’s = .11, p’s < .001; Tables 25, 27, 29). This relationship varied significantly across individuals (estimates of variance = .03, p’s < .001), with some participants exhibiting a greater association between high demand and NA in comparison to others.

3.4.1.2 Sleep Characteristics x Work Demand Effects

Sleep midpoint, sleep duration, and sleep efficiency were not associated with PA or NA (p’s > .05). In addition, the three sleep characteristics did not significantly moderate the effects of
work demand on either PA or NA (p’s >.05, Tables 24-29). There were statistically significant individual differences in the sleep duration*demand effect on NA, and the sleep efficiency*demand on PA, but the variabilities were negligible (estimates of variance =.00). There were no significant individual differences in the other interaction effects between sleep characteristics and work demand on PA or NA (p’s >.05).

### 3.4.2 The Effects of Sleep Characteristics and Work Latitude on Affect

#### 3.4.2.1 Work Latitude, PA, and NA

Low work latitude during a given hour was associated with lower levels of PA (β = -.19, p <.001) and greater levels of NA (β = .17, p <.001) during the same hour (Tables 30-35). This relationship between work latitude and PA varied significantly across participants with some participates exhibiting a stronger association than others (estimate of variance =.05, p <.001). There were also individual differences in regard to NA, such that some participants exhibited a greater association between work latitude and NA than others (estimate of variance =.04, p <.001).

#### 3.4.2.2 Sleep Characteristics*Work Latitude Effects

Sleep midpoint, duration, and efficiency were not related to either PA or NA, and the three sleep characteristics did not significantly moderate the effects of work latitude on either PA or NA (p’s >.05; Tables 30-35). There were, however, several individual differences in the interaction effects. There were significant individual differences in the sleep midpoint* latitude effect on both PA and NA (estimates of variance =.01, p’s <.001). Likewise, there were individual differences in the sleep duration* latitude effect on PA (estimate of variance =.01, Wald Z =3.27, p =.001). While there were statistically significant individual differences in the sleep duration*latitude effect
on NA and in the sleep efficiency*latitude effects on PA and NA, the estimated variabilities were negligible (estimates of variance = .00).

3.4.3 The Effects of Sleep Characteristics and Social Conflict on Affect

3.4.3.1 Social Conflict, PA, and NA

Greater social conflict at a given moment predicted lower levels of PA (B’s = -.34 - -.33, p’s <.001) and higher levels of NA (B’s = .35, p’s <.001; Tables 36-41). There were also significant individual differences in these relationships such that some participants exhibited a stronger and others a weaker conflict-PA relationship (estimates of variance = .03-.04, p’s <.001) and conflict-NA relationship (estimate of variance = .02-.03, p’s <.001).

3.4.3.2 Sleep Characteristics*Social Conflict Effects

Sleep midpoint, duration, and efficiency were not associated with either PA or NA, nor did these sleep characteristics modify the effects of social conflict on either PA or NA (p’s >.05; Tables 36-41). There were significant individual differences in the sleep midpoint*social conflict interaction effect on NA (estimate of variance = .01, Wald Z =2.13, p =.033). However, there were no statistically significant or meaningful (estimates of variance = .00) individual differences in the either sleep duration*conflict or sleep efficiency*conflict effects on PA and NA (p’s >.05).
3.4.4 The Effects of Sleep Characteristics and Pleasant Social Interactions on Affect

3.4.4.1 Pleasant Social Interactions, PA and NA

More pleasant social interaction at a given moment predicted higher levels of PA (B’s=.44-.45, p’s <.001) and lower levels of NA (B’s=-.27-.26, p’s <.001; Tables 42-47). There were significant individual differences such that participants exhibited a stronger and others a weaker association between conflict and PA (estimates of variance=.01, p’s <.001), and between conflict and NA (estimates of variance=.01, p’s <.001).

3.4.4.2 Sleep Characteristics*Social Conflict Effects on PA and NA

Sleep midpoint, duration, and efficiency were not related to PA or NA (p’s >.05), nor did these sleep characteristics modify the effects of pleasant interactions on either PA or NA (p’s >.05; Tables 42-47). There were significant individual differences in the effects of the sleep midpoint*pleasant effect on PA (estimate of variance=.01, Wald Z =3.21, p =.001), and on NA (estimate of variance=.01, Wald Z =2.86, p =.004). In addition, there were significant individual differences in the sleep duration*pleasant interaction effect on PA (estimate of variance=.01, Wald Z =4.55, p <.001). While there were statistically significant individual differences in the sleep duration*pleasant interaction effects on NA and in the sleep efficiency*pleasant interaction effects on both PA and NA, the estimated variability for each relationship was negligible (estimates of variance=.00, p’s <.001).
3.4.5 Secondary Analyses for Social Conflict and Pleasant Interactions

Of note, while participants were asked to report on social interactions that occurred up to 60 minutes prior to the electronic diary assessment (EMA), our primary analyses only included social interactions that occurred within 10 minutes of the electronic diary assessment. This allowed us to focus exclusively on the proximal relationships between social interactions and affect. Since it is possible that social interactions may have delayed effects on affect, we conducted secondary analyses including all social interactions reported up to 60 minutes before the EMA. Despite this, we continued to find that while there were effects of social interactions on PA and on NA (p’s <.05), sleep characteristics did not modify these effects (p’s >.05).

3.4.6 Summary

Overall, we found that neither sleep duration, continuity nor timing significantly interacted with daily experiences to influence PA or NA. However, we found various individual differences in how sleep midpoint modified the effects of work latitude and pleasant interactions on both PA and NA, and how sleep midpoint modified the effects of social conflict on NA.

3.5 Exploratory Analyses: Potential Moderators

As reported above, we found that while there were no main effects of sleep midpoint on PA or NA, there were significant individual differences in these associations (p’s <.05). Similarly, we found individual differences in the effects of PA on sleep midpoint and duration (p’s <.05). To
further examine what may contribute to these individual differences, we conducted exploratory analyses to test whether seven participant characteristics (age, sex, race, years of schooling and family income as indicators of socioeconomic status, neuroticism, and chronotype) moderate these effects. We conducted 14 analyses to test for an interaction between sleep midpoint and each of these characteristics on PA and on NA. We then conducted 14 analyses to test for possible interaction effects between PA and these characteristics on both sleep midpoint and sleep duration.

Overall, the analyses revealed no significant interaction effects with the exception of two findings. We found that none of these participant characteristics significantly modified the effects of sleep midpoint on PA or on NA (p’s >.05; data not shown). There were no significant moderating effects of participant characteristics with respect to effects of PA on sleep midpoint (p’s >.05; data not shown). In regard to sleep duration, we found that participant characteristics did not modify the effects of PA (p’s >.05), with the exception of age and years of schooling (p’s <.001).

As shown in Figure 2, we found an interaction of age and PA on sleep duration (B =.04, CI: .01-.07, p =.006). While age and PA were modeled as continuous variables, for illustrative purposes we plotted sleep duration as a function of age group. After experiencing higher PA, older participants tended to sleep more (longer duration) relative to when they experienced lower PA. In contrast, younger participants tended to sleep less on the corresponding night when they experienced higher PA compared to lower PA.
Figure 2 Participant Age Modifies the Effect of Positive Affect on Sleep Duration.

PA refers to person-centered positive affect. While PA was modeled as a continuous variable, for illustrative purposes we depict PA in the following categories: Average (7.74E-03), High (1SD above average, 1.05E-01), and Low (1SD below average, -8.91E-02). Age groups were also depicted categorically as: Average age (42.8 yrs old), Older as 1SD above (60.14 yrs), and Younger as 1SD below (35.44 yrs).

We also found an interaction of years of schooling and PA on sleep duration (B=-.08, CI: -.15 - -.01, p =.024; Figure 3). When experiencing lower levels of PA, those with less years of schooling tended to sleep less in comparison to when they experienced higher levels of PA. However, those with more years of schooling tended to sleep more when experiencing lower PA relative to higher PA.
Figure 3 Years of Schooling Modifies the Effect of Positive Affect on Sleep Duration.

PA refers to person-centered positive affect. While PA was modeled as a continuous variable, for illustrative purposes we depict PA in the following categories: Average (7.74E-03), High (1SD above average, 1.05E-01), and Low (1SD below average, -8.91E-02). Years of schooling is also depicted categorically: Average (17.0 yrs), More Years (1SD above average, 19.8 yrs), and Less Years (1SD below, 14.1 yrs).

In regard to the effects of sleep on affect reactivity, we found additional individual differences in how sleep midpoint and duration interacted with various daily experiences to influence affect. However, based on the lack of significant findings relative to the number of analyses conducted above (2 out of 28), we did not conduct exploratory analyses regarding this last set of individual differences, which would involve testing an additional 49 models.
4.0 Discussion

The aim of the current study was two-fold: first, to examine whether there is a proximal, bi-directional relationship between sleep characteristics and affect, and second, to test whether sleep characteristics on a given night influence people’s next-day affect reactivity to social and work experiences. We found that a greater increase in a person’s positive affect was related to a later shift in their sleep midpoint. In contrast to our hypothesis, we found that neither sleep midpoint, sleep duration, nor sleep efficiency on a given night predicted next day affect. We also found that positive affect during the daytime was unrelated to sleep duration and sleep efficiency, and negative affect was unrelated to all sleep characteristics. While we found that participant’s reported work and social experiences were significantly associated with positive and negative affect, participants’ sleep characteristics the preceding night did not modify these effects. Although testing individual differences in the sleep-affect relationships was not the focus of the current study, our results pointed towards possible individual differences that we further explored.

Our findings suggest no associations between sleep, affect, and affect reactivity, with the exception that PA influenced sleep timing. Overall, these results add to the existing mixed literature. We originally proposed that previous results were mixed in part because studies were limited in study methodology (assessments of sleep and affect). We aimed to address these limitations and, to the best of our knowledge, we are the first study to use a combination of both actigraphy and EMA measures of affect. Comparison of our participant demographics, study design and methods, and conceptual framework to the larger literature on sleep and mood can inform interpretation of our findings.
4.1 Participant Sleep and Affect Characteristics

In the current sample, participants’ sleep characteristics were consistent with previous reports on healthy adults. Here, participants slept an average of 6.8 hours and shifted night-to-night on average by 58 minutes. This is consistent with another study that showed healthy adults without insomnia slept an average 6.6 hours and deviated on average by 53.9 min in their sleep duration (Buysse et al., 2010). We found that participants reported an average bedtime of 11:42PM and wake time of 6:31AM, resulting in an average sleep midpoint of 3:06AM. There was considerable night-to-night variable, with participants’ sleep midpoint standard deviation being 64 minutes. Similarly, Buysse et al. (2010) found their participants had an average 11:26PM bedtime and varied 70.9 minutes night-to-night. In addition, participants generally showed normal sleep efficiency (83.1%, SD= 5.4%, average night-to-night variation = 4.0%), with a cutoff of 85% indicating “good” sleep efficiency. While participants were not screened for sleep disorders, their normal sleep efficiency and body mass index (BMI; 26.8 ± 5.2) suggests it is unlikely participants had sleep-related disorders such as obstructive sleep apnea. Overall, our sample exhibited sleep characteristics similar to that previously observed in healthy adults free of sleep disorders.

Most previous studies of the sleep-affect relationship used various forms of affect measures and scoring metrics, which precludes their direct comparison with our participants’ EMA-reported affect. In our sample, participants tended to rate high on PA and low on NA, with some variability as assessed on hourly intervals throughout the day: participants reported an average PA of 4.0 (average nightly variation =0.24), and an average NA of 1.9 (average nightly variation=.16). Participants were only included in the AHAB study if they were psychiatrically healthy and excluded if taking medication. These affect characteristics are thus consistent with the
demographic of our sample and our findings represent the relationships between sleep and affect that unfold among healthy adults.

We generally found a sufficient amount of within-person variability to test our hypotheses. A medium to large proportion (40.8 - 87.3%) of variance in our sample’s sleep efficiency, sleep timing, PA and NA were attributable to within-person, day-to-day differences. Of note, there was relatively less within-person variability in sleep duration (16.9%). Overall, our study was equipped to test the daily relationships between sleep and affect, with the exception that some results may reflect insufficient variability in sleep duration.

4.2 The Effects of Sleep Characteristics on Affect

We hypothesized that shorter sleep duration, less sleep continuity, and later sleep timing on a given night would predict greater levels of PA and lower levels of NA the following day. However, we found no significant effects of any of these sleep characteristics on PA or NA. We aimed to address the limitations in methodology and study design across previous studies and, to the best of our knowledge, we were the first study to use a combination of both actigraphy and EMA measures of affect. Our findings indicate that sleep characteristics, as measured behaviorally rather than subjectively, do not influence affect on a proximal, day-to-day level.

4.2.1 Sleep Duration

We found an effect of sleep duration on NA that trended towards significance, but the effect size was minimal. Our finding that sleep duration generally did not predict next day affect adds to
previous mixed findings. In contrast to our hypotheses, we found no significant linear or nonlinear relationship between sleep duration and affect. It is possible that the relatively low within-person variability in sleep duration among our sample may have contributed to the null findings. Since previous studies did not report within-person variability in sleep duration, we are unable to determine whether this low variability differentiates the present study from those that reported significant associations.

It is possible that participants in our study did not experience fluctuations in their sleep duration large enough to influence their mood. In contrast to our findings, previous studies have consistently shown that experimental manipulations of sleep duration have next-day effects on mood (e.g., Babson et al., 2010; Baum et al., 2014; Dinges et al., 1997; Haack & Mullington, 2005). However, these manipulations involved either total deprivation or greater sleep restriction than what our participants naturally experienced. For instance, previous studies restricted people’s sleep duration 33-50% below their habitual sleep duration (Dinges et al., 1997; Haack & Mullington, 2005). In such cases, a participant who normally slept 7 hours would be restricted to 4.7 hours in bed. In our study, participants slept an average of 6.8 hrs (SD = 54 min) and had an average daily shift of 57.6 minutes. This suggests that one person may have slept 6.8 hrs one night and 5.8 hrs the next night, which is only an 15% restriction. Thus, we may not have observed an effect of sleep duration on mood because the participants did not naturally fluctuate in their sleep duration enough to experience the degree of sleep restriction needed to influence subjective mood.

While we did not find a day-to-day effect of sleep duration on affect, there may be cumulative effects that we were unable to consider. Previous studies that measured daily affect over several consecutive days of sleep restriction reported that the effects of short sleep duration (i.e., 4-5 hours/night) on mood accumulated over time such that participants continued to report
increases in negative mood and decreases in PA across several days (Dinges et al., 1997; Haack & Mullington, 2005). Such accumulative effects of sleep restriction have been replicated in the larger literatures on cognitive performance and sleep propensity, with evidence that regular sleep restriction over time can have detrimental effects on performance equivalent to a night of complete sleep deprivation (Banks, 2007; Belenky et al., 2003; Van Dongen, Maislin, Mullington, & Dinges, 2003). Taken together, it is possible that single night shifts in sleep duration have negligible effects, but these effects can accumulate over time to have an influence on affect.

4.2.2 Sleep Continuity

We found that sleep efficiency was not related to either PA or NA. This finding is inconsistent with the literature that consistently linked poor subjective sleep quality and poor subjective sleep continuity to higher NA and lower PA (e.g., Brissette & Cohen, 2002; Scott & Judge, 2002; McCrae et al., 2008; de Wild-Hartmann et al., 2013). Our null finding is consistent with one study wherein authors found perceived, but not actigraphy-derived, measures of nighttime awakenings associated with affect (McCrae et al., 2008). Taken together, our findings suggest that unlike the well documented association with perceived sleep disturbances, behavioral measures of sleep continuity are not associated with affect.

Understanding the differences between sleep continuity and subjective sleep quality has implications for the interpretation of our findings. Actigraphy is a tool that, relative to self-report measures, is a more valid instrument to assess sleep patterns. As discussed previously, self-reported sleep characteristics are often only minimally correlated with those quantified by polysomnography (PSG; Kushida et al., 2001; McCall & McCall, 2012; Silva et al., 2007). In contrast, the epoch-by-epoch agreement rates between actigraphy and PSG in detecting sleep are
high, particularly for healthy individuals (> .85 agreement rates; Sadeh & Acebo, 2002; Ancoli-Israel et al., 2003; Jean-Louis et al., 2001; Marino et al., 2013). Thus, actigraphy provides information on behavioral sleep patterns that correspond with objectively defined sleep while self-reported characteristics are distinct measures of perceived sleep experiences.

The correlation between actigraphy and self-reported sleep characteristics varies widely depending on sleep dimension, with some evidence that sleep continuity has the greatest discrepancy between methodologies. For instance, there appears to be a moderate to high correlation between actigraphy-derived and self-report measures of sleep duration and sleep timing (Auger, Varghese, Silber, & Slocumb, 2013; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008; Lockley, Skene, & Arendt, 1999; McCall & McCall, 2012; Tomita et al., 2013). In contrast, there are widely varying correlations between methods for different forms of sleep continuity (e.g., awakenings and time it takes to fall asleep; r’s = .06-.59; Lockley et al., 1999; McCall & McCall, 2012). Thus, consideration of methodological differences and their implications is most important when interpreting findings on sleep continuity and subjective sleep quality.

While actigraphy-derived measures of sleep continuity overlap with subjective sleep quality for several sleep characteristics, the two methodologies represent distinct sleep dimensions. In the current study, we defined sleep continuity as the percentage of time a person is sleeping in comparison to the total amount of time they are in bed, which takes into account nighttime awakenings and time it takes to fall asleep. While sleep quality measures also assess nighttime awakenings and trouble falling asleep, it has been shown that people base their sleep quality rating on their mood and daytime experiences (Harvey, Stinson, Whitaker, Moskovitz, & Virk, 2008). Specifically, when participants were asked to describe their experience of “poor” sleep quality and of “good” sleep quality, participants tended to include descriptions of their motivation to get
up, their tiredness throughout the day, anxiety, worry, and their mood throughout the day (Harvey et al., 2008). In other words, while sleep continuity captures different forms of nighttime sleep disruptions, self-reported sleep quality appears to capture a person’s reappraisal of their sleep experience that is in part influenced by their mood.

Overall, we found that behaviorally defined sleep continuity does not have proximal effects on affect. Taken together with previous literature, our findings suggest that the well documented link between sleep quality and mood is specific to subjective perceptions of sleep, does not extend to behaviorally-determined sleep continuity, and in part reflects the confounding correlation between mood with sleep quality assessments.

4.2.3 Sleep Timing

We found that day-to-day changes in sleep timing did not significantly predict PA or NA. There is a paucity of studies on the effects of sleep timing on mood. We initially aimed to extend this literature and, to the best of our knowledge, are the first study to examine the effects of naturally occurring, day-to-day sleep timing on both PA and NA. One previous study showed that following days when people slept later, they reported less cheerfulness (Totterdell et al., 1994), and another showed that inducing shifts in sleep timing increased NA and decreased PA (Taub & Berger 1974, 1976). Based on these findings, we originally hypothesized that variability itself in sleep timing would associate with greater NA and lower PA. However, our findings did not support this hypothesis.

Similar to our interpretation of the sleep duration results, it is possible that our null sleep timing results stems in part from either difference in sleep assessment or magnitude of shift in sleep time. For instance, Totterdell et al. (1994) reported their sample had an average sleep onset
at 11:46PM and had larger nightly deviations of 69 minutes, with the latter deviation being larger than what we found in our sample. They examined perceived sleep timing whereas we used actigraphy-derived sleep timing. It is possible that the difference in measurement tool contributed to our inconsistent findings, although there is evidence that the correlations between actigraphy-derived and self-reported sleep timing are moderate to high (r’s =.57-.88; Lockley et al., 1999; McCall & McCall, 2012). On the other hand, experimental sleep studies measured sleep timing objectively and found significant mood changes after either advances or delays in sleep timing (Taub & Berger 1974, 1976). Of note, these studies induced 2-4 hour shifts in sleep time, which are significantly larger than what our participants experienced night-to-night (average deviation of 39.6 minutes). Given the paucity of literature, it is unclear if these methodological or empirical differences contribute to our inconsistent findings.

Our original hypothesis was based on a larger literature documenting an association between evening chronotype, late sleep timing, and depressed mood (e.g, Biss & Hasher, 2012; Hasler et al., 2010; Hasler et al., 2012; Hidalgo et al., 2009; Levandovski et al., 2011). People with evening chronotypes, or a preference for late sleep and wake times, can experience circadian disruption from having to regularly shift between their preferred sleep time and actual sleep time enforced by obligations (Roenneberg et al 2003). This shift in sleep time has been linked to depression. For instance, a previous population-based study found participants who had >2hr discrepancy in sleep on workdays and non-workdays were more likely to be depressed than the rest of the participants (Levandovski et al., 2011). This finding parallels results from aforementioned studies that showed a 2+ hour shift in sleep time associated with mood (Taub & Berger 1974, 1976) and suggests the effects of sleep timing on mood occur in the context of relatively large shifts in sleep time.
Based on previous literature, we expected to find that greater shifts in sleep timing would similarly associate with greater negative affect and lower positive affect. We found that our participants on average varied by 64 min in sleep timing, and there were no significant effects of sleep timing on either PA or NA. Our findings suggest that the circadian system is resilient to relatively smaller, daily variations in sleep time.

4.3 The Effects of PA and NA on Sleep Characteristics

Our second aim was to test whether daily affect would influence nighttime sleep characteristics on the same day. Relative to work on the day-to-day effects of sleep characteristics on affect, fewer studies that have examined effects of affect on subsequent sleep. We aimed to extend this literature and, to the best of our knowledge, this is the first study to test the influence of either PA or NA on sleep timing. We found that greater PA on a given day predicted later sleep time that night. In contrast, we found no significant effect of PA on sleep duration or sleep continuity, and we found no significant effect of NA on any of the sleep characteristics.

We found that greater daytime levels of PA predicted later sleep timing. Our finding adds to the literature given that only one study previously tested the day-to-day effects of PA on sleep timing and found no significant effect (Totterdell et al., 1994). We originally hypothesized that greater NA, rather than PA, would predict later sleep timing. This hypothesis was based on existing evidence that depression is associated with later sleep time and a preference for late sleep time (Biss & Hasher, 2012; Hasler, Allen, Sbarra, Bootzin, & Bernert, 2010; Hasler et al., 2012; Hidalgo et al., 2009; Levandovski et al., 2011). Our finding suggests a different interpretation. As discussed below, we found that positive social interactions and lower work strain predicted higher
momentary measures of PA. While untested here, it is possible that the inverse relationship may
be true: higher levels of PA may lead to more engagement in positive social interactions and
activities. In addition, this positive engagement may extend into the later evening and delay sleep
time. Further studies are warranted to examine whether pre-sleep activities and the late timing of
activities may contribute to the association between daytime PA and later sleep time.

Aside from the PA-sleep timing relationship, we found no significant effects of either PA
or NA on sleep characteristics. Previous studies that tested these day-to-day associations showed
inconsistent results, but we originally predicted that changes in daytime mood can influence sleep
via both physiological arousal and cognitive forms of hyperarousal (Riemann et al., 2010; Tang &
Harvey 2004; Zoccola et al., 2009). For instance, one experimental study found that after
participants were induced to have either cognitive arousal (cognitive activity and anxiety) or
physiological arousal, they reported longer sleep onset latency and shorter sleep duration (Tang &
Harvey 2004). Another study found that after failing a cognitive task before bed, participants
reported greater NA and had more difficulty falling asleep, more nighttime awakenings, and slept
less compared to their baseline characteristics (Vandekerckhove et al., 2011). These participants
reported an average PANAS NA score of 2.10 (± 0.78) after experiencing failure. It is thus
surprising that we did not find similar effects of NA on sleep in our sample given that our
participants reported a similar range of NA (1.9 ± 0.70). However, we tested the effects of affect
as an average daytime level. It is possible that affect at bedtime, rather than overall daytime affect,
influences sleep characteristics.
4.4 Individual Differences in the Sleep-Affect Relationship

While there were no statistically significant effects of sleep characteristics on affect in the total sample, we consistently found individual differences in the relationships between sleep midpoint and affect. Specifically, some participants showed a stronger decline in PA following a delay in sleep midpoint while others showed less change. And, some participants showed a stronger increase in NA following a delay in sleep midpoint than others. This suggests that some people are more likely to experience mood-related consequences of shifts in sleep time relative to others. As noted earlier, we investigated several possible participant characteristics that may modify the effects of sleep timing on affect and thereby contribute to these individual differences. However, we did not find any significant effects of chronotype or other characteristics. Future studies are warranted to identify other markers that distinguish these individual differences.

Regarding the effects of affect on sleep characteristics, we found individual differences in the relationships between PA, sleep midpoint and sleep duration. Specifically, following days of higher PA, some people showed greater delays in their sleep midpoint whereas some showed greater advances in their sleep midpoint. We found similar individual differences in the effects of PA on sleep duration. While some participants slept longer following days of higher PA, others slept less. These findings suggest there are individual traits or situational differences that modify how their mood influences their sleep.

We again tested whether seven participant characteristics moderated the effects of PA on sleep midpoint and duration. We found that none of the participant characteristics significantly modified the effects of PA on sleep midpoint. In regard to sleep duration, we found that both age and years of schooling modified the effects of PA on sleep duration. However, these minimal findings need to be interpreted with caution due to the relative number of analyses we conducted.
4.5 Sleep, Daily Experiences, and Affect Reactivity

We hypothesized that sleep influences affect reactivity such that sleep characteristics would moderate the effects of various daily experiences on affect. Consistent with the previous literature, we found that when participants had more social conflict, higher work demands, and lower work control, they subsequently reported higher levels of NA and lower PA (e.g. Mroczek & Almeida, 2004; Sliwinski, Almeida, Smyth, & Stawski, 2009; Stawski et al., 2008). And, when participants reported more pleasant social interactions, they reported higher levels of PA and lower NA. Contrary to our hypothesis, we did not find any significant interaction between sleep characteristics and daily experiences.

One interpretation of our findings is that small, daily fluctuations in people’s sleep characteristics do not impact affect reactivity. Evidence that sleep duration plays a role in affect reactivity is based largely on findings from total sleep deprivation studies (Babson, Trainor, Feldner, & Blumenthal, 2010; Baum et al., 2014; Franzen et al., 2008; Talbot, McGlinchey, Kaplan, Dahl, & Harvey, 2010). One field study on medical residents extended these experimental findings and showed that restricted sleep and more nighttime awakenings led to more NA following negative work experiences in comparison to days they slept more (Zohar et al., 2005). Based on this evidence, we predicted that our participants would similarly show changes in their affect reactivity following shorter sleep, less continuity, and variable sleep times. Our null results may in part be due to differences in sleep duration given that our participants generally slept more than those in the previous study. Taken together, our findings suggest that there may be graded effects of sleep disruptions on affect reactivity and that the emotion regulation system may be tolerant to smaller, daily fluctuations in sleep.
One strength of the current study involves the use of thorough measures and statistical modeling of affect levels throughout the day. Specifically, we collected repeated measures of affect at regular intervals throughout the day and controlled for time of assessment in order to account for the diurnal rhythm in affect. This approach addressed limitations in the aforementioned study by Zohar and colleagues (2005). While the previous study showed an association between sleep restriction and PA levels throughout the day, the authors assessed affect at random intervals and did not consider diurnal influences. In contrast, we did not find any significant sleep duration-PA association, and this may be because we controlled for the confounding diurnal rhythm of PA.

4.5.1 Individual Differences in the Effects of Sleep on Affect Reactivity

We found several individual differences in the relationships between sleep characteristics and affect reactivity. Specifically, there were individual differences in the extent that sleep midpoint may have modified the effects of work latitude and pleasant interactions on both PA and NA, and in the extent that sleep midpoint may have modified the effects of social conflict on NA. Additionally, there was evidence of individual differences in how sleep duration modified the effects of work latitude and pleasant interactions on PA. Overall, these findings suggest there may be other factors that influence how a person’s sleep influences his/her emotional response to daily experiences.

Individual differences in emotion regulation skills may contribute to the observed differences in how sleep duration and sleep timing interacted with daily experiences across participants. People differ in their use of strategies to regulate their emotions, with one form of emotion regulation strategy being cognitive reappraisal. Cognitive reappraisal is an antecedent-focused form of emotion regulation by which individuals change the meaning of emotional stimuli
and thereby intervene with the development of their emotional response (Gross & John, 2003; John & Gross, 2004). When confronted with high levels of stress, greater cognitive reappraisal capability associates with lower depressive symptoms (Troy, Wilhelm, Shallcross, & Mauss, 2010); reappraisal skills may thus protect individuals from developing negative affect when faced with stressful or unpleasant experiences. For instance, sleep deprivation has been shown to alter the neural circuitry underlying emotion regulation and exaggerate people’s emotional responses to various laboratory stimuli that model pleasant and unpleasant negative experiences (Franzen et al., 2009; Franzen et al., 2008; J. D. Minkel et al., 2012; Walker & van Der Helm, 2009; Yoo et al., 2007). We would expect that people with more emotion regulation skills at baseline would exhibit less emotional reactivity to daily experiences following sleep disruptions in comparison to individuals who have less skills. Future studies are warranted to test this hypothesis.
5.0 Conclusion

The current study aimed to examine whether there is a proximal, bi-directional relationship between sleep characteristics and affect and to test whether sleep characteristics on a given night influence people’s next-day affect reactivity to daily experiences. To the best of our knowledge, this is the first study to exploit both actigraphy and EMA measures of affect. We found that greater PA predicted a delay in sleep midpoint, but there were no other effects of sleep characteristics on affect or, conversely, any effects of affect on sleep characteristics. These findings are surprising given the well-documented relationship between sleep characteristics and mood. In the context of studies that examine the daily, proximal sleep-affect relationships, our findings add to a body of mixed results. While there was no significant effect of sleep characteristics on affect reactivity, we documented several, albeit yet largely unexplained, individual differences across participants in how sleep characteristics interacted with daily experiences to influence affect. Taken together, our findings suggest that, in contrast to perceived sleep experiences, day-to-day fluctuations in behavioral sleep patterns generally do not associate with subsequent affective experience. It remains possible, however, that there may be graded and cumulative effects of sleep disruptions on affect and affect reactivity that are not observed in the context of small, daily fluctuations in sleep characteristics.
Appendix A Tables
<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Participant N (Mean Age, SD)</th>
<th>Period</th>
<th>Sleep Measure(s)</th>
<th>Mood/Affect Measure</th>
<th>The Effect of Sleep Characteristics on Affect</th>
</tr>
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<tr>
<td>Totterdell et al.</td>
<td>1994</td>
<td>30 (M =31.6, range =20-59)</td>
<td>2 weeks</td>
<td>A.M. Report</td>
<td>VAS every 2 hours</td>
<td>▼Continuity → ▼Cheerfulness</td>
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<td>▼SQ → ▼Cheerfulness</td>
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<td>Later Sleep Onset → ▼Cheerfulness</td>
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<tr>
<td>Brissette &amp; Cohen</td>
<td>2002</td>
<td>47 (M =34.0, SD =10.7)</td>
<td>7 days</td>
<td>P.M. Report of previous night (Interview)</td>
<td>P.M. Report (Parts of POMS)</td>
<td>▼Duration → ↑NA, PA N.S.</td>
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<td></td>
<td></td>
<td>▼Continuity → ↑NA, PA N.S.</td>
</tr>
<tr>
<td>Scott &amp; Judge</td>
<td>2006</td>
<td>51 (M =34.9, SD =11.8)</td>
<td>3 weeks</td>
<td>P.M. Report of previous night (Jenkins)</td>
<td>P.M. Report (PANAS-X)</td>
<td>▼Continuity → ↓PA, ↑NA</td>
</tr>
<tr>
<td>McCrae et al.</td>
<td>2008</td>
<td>116 (M =72.8, SD =7.1)</td>
<td>14 days</td>
<td>A.M. Report &amp; Actigraphy</td>
<td>A.M. Report (PANAS)</td>
<td>▼Continuity → ↓PA ↑NA.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>Continuity (Actigraphy) → PA N.S., NA N.S.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>▼SQ → ↓PA, ↑NA.</td>
</tr>
<tr>
<td>Sonnentag et al.</td>
<td>2008</td>
<td>166 (M =38.6, SD =10.7)</td>
<td>7 days</td>
<td>A.M. Report (1 item from PSQI)</td>
<td>A.M. Report (PANAS)</td>
<td>▼Duration → ↓PA, NA N.S.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td>▼SQ → ↓PA and ↑NA</td>
</tr>
<tr>
<td>Galambos &amp; Dalton</td>
<td>2009</td>
<td>191 (M =18.4, SD =0.5)</td>
<td>2 weeks</td>
<td>P.M. Report of previous night</td>
<td>P.M. Report (PANAS)</td>
<td>▼Duration → ↑NA, PA N.S.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>▼SQ → ↓PA, ↑NA</td>
</tr>
<tr>
<td>Wild-Hartmann et al.</td>
<td>2013</td>
<td>551 Women (M =27.8, SD =7.9)</td>
<td>5 days</td>
<td>A.M. Report</td>
<td>EMA, 10x/day, 1x/90min bin</td>
<td>▼Duration → ↓PA, NA N.S.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>▼Continuity → ↓PA, ↑NA</td>
</tr>
<tr>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>▼SQ → ↓PA, ↑NA</td>
</tr>
</tbody>
</table>
Legend: Studies shown assessed the temporal relationship between sleep characteristics and affect. Significant results (p < .05) are shown with arrows representing the directionality of the effect. EMA refers to ecological momentary assessment, PA, Positive Affect, NA, Negative Affect, Duration, Sleep Duration; Continuity, Sleep Continuity; SQ, Sleep Quality. N.S. refers to non-significant results (p > .05). Not Tested indicates the study did not report on the association.

<table>
<thead>
<tr>
<th>8</th>
<th>Kalmbach et al.</th>
<th>2014</th>
<th>171 Women (M =20.1, SD =3.3)</th>
<th>2 weeks</th>
<th>A.M. Report (3 items from PSQI)</th>
<th>A.M. Report (PANAS-X)</th>
<th>Duration → PA N.S., NA N.S. Continuity → PA N.S., NA N.S. ↓SQ → ↓PA, NA N.S.</th>
</tr>
</thead>
<tbody>
<tr>
<td>9</td>
<td>Wrzus et al.</td>
<td>2014</td>
<td>397 (M =39.9, SD =20.5)</td>
<td>9+ days</td>
<td>A.M. Report</td>
<td>EMA, 6x/day, 1x/2hrs</td>
<td>Duration → Quadratic relationship with NA &amp; PA (in participants 20+ yrs old)</td>
</tr>
</tbody>
</table>
Table 2 The Day-to-Day Effects of PA and NA on Sleep Characteristics in Healthy Adults

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Participant N (Mean Age, SD)</th>
<th>Period</th>
<th>Sleep Measure(s)</th>
<th>Mood/Affect Measure</th>
<th>The Effect of Affect on Sleep Characteristic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Totterdell et al.</td>
<td>1994</td>
<td>30 (M =31.6, range: 20-59)</td>
<td>2 weeks</td>
<td>A.M. Report</td>
<td>VAS every 2 hours</td>
<td>Cheerfulness → Continuity N.S., Sleep Onset N.S., SQ N.S.</td>
</tr>
<tr>
<td>Brissette &amp; Cohen</td>
<td>2002</td>
<td>47 (M =34.0, SD =10.7)</td>
<td>7 days</td>
<td>P.M. Report of previous night (Interview)</td>
<td>P.M. Report (Parts of POMS)</td>
<td>↑NA → ↓Continuity, Duration N.S.</td>
</tr>
<tr>
<td>Scott &amp; Judge</td>
<td>2006</td>
<td>51 (M =34.9, SD =11.8)</td>
<td>3 weeks</td>
<td>P.M. Report of previous night (Jenkins)</td>
<td>P.M. Report (PANAS-X)</td>
<td>PA → Continuity N.S.</td>
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<tr>
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<td>NA → Continuity N.S.</td>
</tr>
<tr>
<td>McCrae et al.</td>
<td>2008</td>
<td>116 (M =72.8, SD =7.1)</td>
<td>14 days</td>
<td>A.M. Report &amp; Actigraphy</td>
<td>A.M. Report (PANAS)</td>
<td>Not Tested</td>
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<td>A.M. Report (PANAS)</td>
<td>Not Tested</td>
</tr>
<tr>
<td>Galambos &amp; Dalton</td>
<td>2009</td>
<td>191 (M =18.4, SD =0.5)</td>
<td>2 weeks</td>
<td>P.M. Report of previous night</td>
<td>P.M. Report (PANAS)</td>
<td>↓PA → Duration N.S., ↓SQ</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>NA → Duration N.S., SQ N.S.</td>
</tr>
<tr>
<td>Wild-Hartmann et al.</td>
<td>2013</td>
<td>551 Women (M =27.8, SD =7.9)</td>
<td>5 days</td>
<td>A.M. Report</td>
<td>EMA, 10x/day, 1x/90min bin</td>
<td>↓PA → Duration N.S., Continuity N.S., ↓SQ</td>
</tr>
<tr>
<td></td>
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<td></td>
<td></td>
<td></td>
<td>NA → Duration N.S., Continuity N.S., SQ N.S.</td>
</tr>
<tr>
<td>Kalmbach et al.</td>
<td>2014</td>
<td>171 Women (M =20.1, SD =3.3)</td>
<td>2 weeks</td>
<td>A.M. Report (3 items from PSQI)</td>
<td>A.M. Report (PANAS-X)</td>
<td>↓PA → ↓Duration, ↓Continuity, ↓SQ</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>↑NA → ↓Duration, ↓Continuity, ↓SQ</td>
</tr>
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<td>Not Tested</td>
</tr>
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</table>
Legend: Studies shown assessed the temporal relationship between sleep characteristics and affect. Significant results (p < .05) are shown with arrows representing the directionality of the effect. EMA refers to ecological momentary assessment, PA, Positive Affect, NA, Negative Affect, Duration, Sleep Duration; Continuity, Sleep Continuity; SQ, Sleep Quality. N.S. refers to non-significant results (p > .05). Not Tested indicates the study did not report on the association.
Table 3 Study Variables Organized By Levels In Model

<table>
<thead>
<tr>
<th>Level 3: Moment-to-Moment, Within-Day</th>
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</thead>
<tbody>
<tr>
<td>Positive Affect (PA)</td>
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<tr>
<td>Negative Affect (NA)</td>
</tr>
<tr>
<td>Work Strain</td>
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<tr>
<td>Negative Social Interaction</td>
</tr>
<tr>
<td>Positive Social Interaction</td>
</tr>
<tr>
<td>Time of Assessment</td>
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</table>

<table>
<thead>
<tr>
<th>Level 2: Day-to-Day, Within-Person</th>
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<tbody>
<tr>
<td>PA (Daily Average)</td>
</tr>
<tr>
<td>NA (Daily Average)</td>
</tr>
<tr>
<td>Sleep Duration</td>
</tr>
<tr>
<td>Sleep Continuity</td>
</tr>
<tr>
<td>Sleep Timing</td>
</tr>
<tr>
<td>Day of Assessment</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Level 1: Between-Person</th>
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</thead>
<tbody>
<tr>
<td>Age</td>
</tr>
<tr>
<td>Sex</td>
</tr>
<tr>
<td>Race</td>
</tr>
<tr>
<td>Sleep Duration (Average)</td>
</tr>
<tr>
<td>Sleep Continuity (Average)</td>
</tr>
<tr>
<td>Sleep Timing (Average)</td>
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</tbody>
</table>
Table 4 Participant Characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Mean (SD) or %</th>
<th>Correlations with Sleep Characteristics</th>
<th></th>
<th></th>
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</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Duration</td>
<td>Midpoint</td>
<td>Efficiency</td>
<td></td>
</tr>
<tr>
<td>Demographics</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>42.7 (7.3)</td>
<td>-.01</td>
<td>-.17**</td>
<td>-.03</td>
<td></td>
</tr>
<tr>
<td>Sex</td>
<td>47.1% Male</td>
<td>.18**</td>
<td>-.10*</td>
<td>-.17**</td>
<td></td>
</tr>
<tr>
<td>Race</td>
<td>81.8% White</td>
<td>-.08</td>
<td>.06</td>
<td>-.12*</td>
<td></td>
</tr>
<tr>
<td>Education (yrs)</td>
<td>17.0 (2.9)</td>
<td>.11*</td>
<td>-.05</td>
<td>.10*</td>
<td></td>
</tr>
<tr>
<td>Family Income</td>
<td>17.9% &gt;110,000</td>
<td></td>
<td></td>
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<tr>
<td>Employment Status</td>
<td>89.6% Full-time</td>
<td>-.00</td>
<td>.08</td>
<td>-.01</td>
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<tr>
<td>Marital Status</td>
<td>62.9% Married</td>
<td>-.07</td>
<td>.23**</td>
<td>-.18**</td>
<td></td>
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<tr>
<td>Average Sleep Characteristics</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>Chronotype</td>
<td>39.3 (7.1)</td>
<td>.08</td>
<td>-.54**</td>
<td>.01</td>
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</tr>
<tr>
<td>Bed Time</td>
<td>11:42PM (1hr 12min)</td>
<td>-.46**</td>
<td>.93**</td>
<td>-.16**</td>
<td></td>
</tr>
<tr>
<td>Wake Time</td>
<td>6:31AM (1hr 8 min)</td>
<td>.31**</td>
<td>.92**</td>
<td>.01</td>
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<tr>
<td>Sleep Midpoint</td>
<td>3:06AM (1hr 4 min)</td>
<td>-.09*</td>
<td>--</td>
<td>-.09</td>
<td></td>
</tr>
<tr>
<td>Sleep Duration</td>
<td>6.8 hrs (54 min)</td>
<td>--</td>
<td>-.09*</td>
<td>.22**</td>
<td></td>
</tr>
<tr>
<td>Sleep Efficiency (%)</td>
<td>83.1 (5.4)</td>
<td>.22**</td>
<td>-.09</td>
<td>--</td>
<td></td>
</tr>
<tr>
<td>PSQI Total</td>
<td>5.1 (2.7)</td>
<td>-.19**</td>
<td>.06</td>
<td>-.13**</td>
<td></td>
</tr>
<tr>
<td>Baseline Depression &amp; Average Levels of Affect</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CES-D Total</td>
<td>8.5 (7.9)</td>
<td>.00</td>
<td>.09</td>
<td>-.03</td>
<td></td>
</tr>
<tr>
<td>Positive Affect</td>
<td>4.0 (0.7)</td>
<td>-.01</td>
<td>-.01</td>
<td>-.06</td>
<td></td>
</tr>
<tr>
<td>Negative Affect</td>
<td>1.9 (0.7)</td>
<td>.07</td>
<td>.01</td>
<td>-.07</td>
<td></td>
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<tr>
<td>Health Behaviors</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Activity (kilocal/day)</td>
<td>2782.0 (2096.7)</td>
<td>.00</td>
<td>-.14**</td>
<td>-.14**</td>
<td></td>
</tr>
<tr>
<td>Alcohol Intake (drinks/wk)</td>
<td>3.1 (4.5)</td>
<td>-.03</td>
<td>.08</td>
<td>-.03</td>
<td></td>
</tr>
<tr>
<td>Smoking Status</td>
<td>13.5% Current Smoker</td>
<td>-.06</td>
<td>.22**</td>
<td>-.12*</td>
<td></td>
</tr>
</tbody>
</table>

Legend: Correlations refer to Pearson bivariate analyses, except where indicated. ¹Point biserial correlations conducted with the described category of each variable serving as the comparison group.  *p < .05, **p < .01
Table 5 The Association between Covariates and Positive Affect

| Fixed Effects |  |  |  |  |  |  |
|---------------|---------------|------------------|------------------|------------------|------------------|
|               | B             | 95% CI           | 95% CI           | SE               | df   | t   | p   |
| Level 1: Within-Person, Daily |               | Lower Limit      | Upper Limit      |                  |      |     |     |
| Workday Status | .09           | .05              | .14              | .02              | 1229.43 | 4.28 | <.001|
| Level 2: Between-Person |               |                  |                  |                  |      |     |     |
| Intercept     | 3.88          | 3.44             | 4.32             | .23              | 442.56 | 17.25 | <.001|
| Age           | .00           | -.01             | .01              | .00              | 442.04 | .62  | .534 |
| Sex           | -.00          | -.14             | .13              | .07              | 441.50 | -.05 | .957 |
| Race          | .12           | -.06             | .30              | .09              | 442.44 | 1.33 | .185 |
| Alcohol       | .00           | -.01             | .02              | .01              | 443.30 | .35  | .729 |
| Smoking Status| -.17          | -.36             | .02              | .10              | 443.44 | -1.72| .086 |
| Physical Activity | 1.39E-7     | -3.09E-5         | 3.12E-5          | 1.58E-5          | 442.19 | .01  | .993 |
| Baseline Depression | -.03         | -.04             | -.02             | .00              | 443.10 | -5.99| <.001|

<table>
<thead>
<tr>
<th>Random Effects</th>
<th>Estimate</th>
<th>95% CI</th>
<th>95% CI</th>
<th>SE</th>
<th>Wald Z</th>
<th>p</th>
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<tbody>
<tr>
<td></td>
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<td>Lower Limit</td>
<td>Upper Limit</td>
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<tr>
<td>Residual</td>
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<td>.13</td>
<td>.15</td>
<td>.01</td>
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<tr>
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<td>.45</td>
<td>.39</td>
<td>.52</td>
<td>.03</td>
<td>13.64</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of affect assessment.
Table 6 The Association between Covariates and Negative Affect

<table>
<thead>
<tr>
<th>Fixed Effects</th>
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<tr>
<td></td>
<td>B</td>
<td>95% CI</td>
<td>95% CI</td>
<td>SE</td>
<td>df</td>
<td>t</td>
<td>p</td>
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</tr>
<tr>
<td>Level 1: Within-Person, Daily</td>
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<td></td>
<td></td>
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<tr>
<td>Workday Status</td>
<td>-.06</td>
<td>-.09</td>
<td>-.03</td>
<td>.02</td>
<td>1221.62</td>
<td>-4.02</td>
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<tr>
<td>Level 2: Between-Person</td>
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<td></td>
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<tr>
<td>Intercept</td>
<td>1.64</td>
<td>1.22</td>
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<td>439.07</td>
<td>7.57</td>
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<tr>
<td>Age</td>
<td>.00</td>
<td>-.01</td>
<td>.01</td>
<td>.00</td>
<td>438.79</td>
<td>.70</td>
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<td>Sex</td>
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<td>-.16</td>
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<td>Race</td>
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<td>.09</td>
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<tr>
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<td>.01</td>
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<td>.95</td>
<td>.341</td>
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<td>-.01</td>
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<td>.09</td>
<td>439.48</td>
<td>1.81</td>
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<td>Physical Activity</td>
<td>-1.40E-5</td>
<td>-4.39E-5</td>
<td>1.60E-5</td>
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<td>-.92</td>
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<td>Baseline Depression</td>
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<td>.02</td>
<td>.04</td>
<td>.00</td>
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<td>95% CI</td>
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<tr>
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<td>.07</td>
<td>.08</td>
<td>.00</td>
<td>24.66</td>
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<td>.50</td>
<td>.03</td>
<td>14.14</td>
<td>&lt;.001</td>
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</tbody>
</table>

Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of affect assessment.
Table 7 Sleep Midpoint Does Not Predict Positive Affect

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Work Day Status indicates if the participant was working on the day of affect assessment. Midpoint Centered refers to person-centered sleep midpoint and represents day-to-day variation.
Table 8 Sleep Midpoint Does Not Predict Negative Affect

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Work Day Status indicates if the participant was working on the day of affect assessment. Midpoint Centered refers to person-centered sleep midpoint and represents day-to-day variation.
Table 9 Sleep Duration Does Not Predict Positive Affect

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Random Effects

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of affect assessment. Duration Centered refers to person-centered sleep duration and represents day-to-day variation.
Table 10 The Association between Sleep Duration and Negative Affect

| Fixed Effects |  |  |  |  |  |  |
|---------------|----------------|----------------|----------------|----------------|---------|
|               | B  | 95% CI Lower Limit | 95% CI Upper Limit | SE  | df   | t    | p   |
| Level 1: Within-Person, Daily |          |                  |                  |      |      |      |     |
| Workday Status | -.07 | -.10 | -.04 | .02 | 1222.13 | -4.45 | <.001 |
| Duration Centered | .01 | .00 | .03 | .01 | 218.94 | 1.99 | .047 |
| Level 2: Between-Person |          |                  |                  |      |      |      |     |
| Intercept      | 1.26 | .60 | 1.92 | .33 | 444.14 | 3.77 | <.001 |
| Age            | .00  | -.01 | .01 | .00 | 437.37 | .72 | .470 |
| Sex            | -.06 | -.19 | .08 | .07 | 437.65 | -.85 | .398 |
| Race           | -.06 | -.23 | .11 | .09 | 438.93 | -.66 | .513 |
| Alcohol        | .01  | -.01 | .02 | .01 | 438.06 | .99 | .321 |
| Smoking Status | .17  | -.01 | .35 | .09 | 438.07 | 1.82 | .070 |
| Physical Activity | -1.48E-5 | -4.47E-5 | 1.52E-5 | 1.52E-5 | 437.54 | -.97 | .334 |
| Baseline Depression | .03 | .02 | .04 | .00 | 438.76 | 6.86 | <.001 |
| Average Duration | .06 | -.02 | .13 | .04 | 446.02 | 1.52 | .130 |

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of affect assessment. Duration Centered refers to person-centered sleep duration and represents day-to-day variation.
Table 11 Sleep Efficiency Does Not Predict Positive Affect

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<th>95% CI Upper Limit</th>
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of affect assessment. Efficiency Centered refers to person-centered sleep efficiency and represents day-to-day variation.
Table 12 Sleep Efficiency Does Not Predict Negative Affect

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<th>95% CI Upper Limit</th>
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of affect assessment. Efficiency Centered refers to person-centered sleep efficiency and represents day-to-day variation.
Table 13 The Association between Covariates and Sleep Midpoint

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Midpoint refers to the participant’s sleep midpoint the preceding night.
Table 14 The Association between Covariates and Sleep Duration

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Duration refers to the participant’s sleep duration the preceding night.
Table 15 The Association between Covariates and Sleep Efficiency

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Efficiency refers to the participant’s sleep efficiency the preceding night.
### Table 16 Higher Levels of Positive Affect Predicts Later Sleep Midpoint

#### Fixed Effects

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#### Random Effects

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Legend:  Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Midpoint refers to the participant’s sleep midpoint the preceding night; PA, Positive Affect; Centered PA, Person-centered positive affect.
Table 17 Negative Affect Does Not Predict Sleep Midpoint

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Legend:  Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Midpoint refers to the participant’s sleep midpoint the preceding night; NA, Negative Affect; Centered NA, Person-centered negative affect.
Table 18 Positive Affect Does Not Predict Sleep Duration

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Duration refers to the participant’s sleep duration the preceding night; PA, Positive Affect; Centered PA, Person-centered positive affect.
### Table 19 Negative Affect Does Not Predict Sleep Duration

#### Fixed Effects

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#### Random Effects

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Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item.

Workday Status indicates if the participant was working on the day of sleep assessment. Previous Duration refers to the participant’s sleep duration the preceding night; NA, Negative Affect; Centered NA, Person-centered negative affect.
Table 20 Positive Affect Does Not Predict Sleep Efficiency

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Random Effects

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Efficiency refers to the participant’s sleep efficiency the preceding night; PA, Positive Affect; Centered PA, Person-centered positive affect.
Table 21 Negative Affect Does Not Predict Sleep Efficiency

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Previous Efficiency refers to the participant’s sleep efficiency the preceding night; NA, Negative Affect; Centered NA, Person-centered negative affect.
Table 22 The Association between Covariates and Hourly Assessments of Positive Affect

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment.
Table 23 The Association between Covariates and Hourly Assessments of Negative Affect

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment.
Table 24 Sleep Midpoint Does Not Moderate the Effects of Hourly Work Demand on Positive Affect

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Centered Midpoint refers to person-centered sleep midpoint on a given night. Work Demand refers to a dichotomous variable with 0 = low demand, 1 = high demand. Cen Midpoint* Demand refers to the interaction between grand-mean centered Centered Midpoint by Demand.
Table 25 Sleep Midpoint Does Not Moderate the Effects of Hourly Work Demand on Negative Affect

<table>
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Centered Midpoint refers to person-centered sleep midpoint on a given night. Work Demand refers to a dichotomous variable with 0 = low demand, 1 = high demand. Cen Midpoint* Demand refers to the interaction between grand-mean centered Centered Midpoint by Demand.
### Table 26 Sleep Duration Does Not Moderate the Effects of Hourly Work Demand on Positive Affect

**Fixed Effects**

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**Random Effects**

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Centered Duration refers to person-centered sleep midpoint on a given night. Work Demand refers to a dichotomous variable with 0 = low demand, 1 = high demand. Cen Duration*Demand refers to the interaction between grand-mean centered Centered Duration by Demand.
### Table 27 Sleep Duration Does Not Moderate the Effects of Hourly Work Demand on Hourly NA

#### Fixed Effects

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Table 28 Sleep Efficiency Does Not Moderate the Effects of Hourly Work Demand on Hourly PA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Centered Efficiency refers to person-centered sleep midpoint on a given night. Work Demand refers to a dichotomous variable with 0 = low demand, 1 = high demand. Cen Efficiency* Demand refers to the interaction between grand-mean centered Centered Efficiency by Demand.
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Work Demand refers to a dichotomous variable with 0 = low demand, 1 = high demand. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency * Demand refers to the interaction between grand-mean centered Centered Efficiency by Demand.
Table 30 Sleep Midpoint Does Not Moderate the Effects of Hourly Work Latitude on Hourly PA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Work Latitude refers to a dichotomous variable with 0 = high latitude, 1 = low latitude. Centered Midpoint refers to person-centered sleep midpoint on a given night. Cen Midpoint*Latitude refers to the interaction between grand-mean centered Centered Midpoint by Latitude.
Table 31 Sleep Midpoint Does Not Moderate the Effects of Hourly Work Latitude on Hourly NA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Work Latitude refers to a dichotomous variable with 0 = high latitude, 1 = low latitude. Centered Midpoint refers to person-centered sleep midpoint on a given night. Cen Midpoint*Latitude refers to the interaction between grand-mean centered Centered Midpoint by Latitude.
### Table 32 Sleep Duration Does Not Moderate the Effects of Hourly Work Latitude on Hourly PA

#### Fixed Effects

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Work Latitude refers to a dichotomous variable with 0 = high latitude, 1 = low latitude. Centered Duration refers to person-centered sleep midpoint on a given night. Cen Duration*Demand refers to the interaction between grand-mean centered Centered Duration by Latitude.
Table 33 Sleep Duration Does Not Moderate the Effects of Hourly Work Latitude on Hourly NA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Work Latitude refers to a dichotomous variable with 0 = high latitude, 1 = low latitude. Centered Duration refers to person-centered sleep midpoint on a given night. Cen Duration * Latitude refers to the interaction between grand-mean centered Centered Duration by Latitude.
Table 34 Sleep Efficiency Does Not Moderate the Effects of Hourly Work Latitude on Hourly PA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Work Latitude refers to a dichotomous variable with 0 = high latitude, 1 = low latitude. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency* Latitude refers to the interaction between grand-mean centered Centered Efficiency by Latitude.
### Table 35: Sleep Efficiency Does Not Moderate the Effects of Hourly Work Latitude on Hourly NA

#### Fixed Effects

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#### Random Effects

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Work Latitude refers to a dichotomous variable with 0 = high latitude, 1 = low latitude. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency * Latitude refers to the interaction between grand-mean centered Efficiency by Latitude.
Table 36 Sleep Midpoint Does Not Moderate the Effects of Hourly Social Conflict on Hourly PA

**Fixed Effects**

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**Random Effects**

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Centered Midpoint refers to person-centered sleep midpoint on a given night. Cen Midpoint*Cen Conflict refers to the interaction between grand-mean centered Centered Midpoint by person-centered Conflict.
Table 37 Sleep Midpoint Does Not Moderate the Effects of Hourly Social Conflict on Hourly NA

### Fixed Effects

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### Level 2: Within-Person, Daily

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### Level 3: Between-Person

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### Random Effects

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<td>.01</td>
<td>.00</td>
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Centered Midpoint refers to person-centered sleep midpoint on a given night. Cen Midpoint*Cen Conflict refers to the interaction between grand-mean centered Centered Midpoint by person-centered Conflict.
Table 38 Sleep Duration Does Not Moderate the Effects of Hourly Social Conflict on Hourly PA

<table>
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<tr>
<th>Fixed Effects</th>
<th>B</th>
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<th>95% CI</th>
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Centered Duration refers to person-centered sleep midpoint on a given night. Cen Duration*Cen Conflict refers to the interaction between grand-mean centered Centered Duration by person-centered Conflict.
Table 39 Sleep Duration Does Not Moderate the Effects of Hourly Social Conflict on Hourly NA

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<th>95% CI Upper Limit</th>
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<th>Wald Z</th>
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<td>3.97</td>
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</table>
Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Centered Duration refers to person-centered sleep midpoint on a given night. Cen Duration*Cen Latitude refers to the interaction between grand-mean centered Centered Duration by person-centered Latitude.
Table 40 Sleep Efficiency Does Not Moderate the Effects of Hourly Social Conflict on Hourly PA

**Fixed Effects**

<table>
<thead>
<tr>
<th></th>
<th>B</th>
<th>95% CI Lower Limit</th>
<th>95% CI Upper Limit</th>
<th>SE</th>
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**Random Effects**

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency*Cen Conflict refers to the interaction between grand-mean centered Centered Efficiency by person-centered Conflict.
Table 41 Sleep Efficiency Does Not Moderate the Effects of Hourly Social Conflict on Hourly NA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency *Cen Conflict refers to the interaction between grand-mean centered Centered Efficiency by person-centered Conflict.
Table 42 Sleep Midpoint Does Not Moderate the Effects of Hourly Pleasant Interactions on Hourly PA

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Random Effects

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<td>3.21</td>
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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Centered Midpoint refers to person-centered sleep midpoint on a given night. Cen Midpoint*Cen Inter refers to the interaction between grand-mean centered Centered Midpoint by person-centered Pleasant Interaction.
Table 43 Sleep Midpoint Does Not Moderate the Effects of Hourly Pleasant Interactions on Hourly NA

### Fixed Effects

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### Random Effects

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Midpoint refers to a participant’s average sleep midpoint across all available monitoring days. Centered Midpoint refers to person-centered sleep midpoint on a given night. Cen Midpoint*Cen Inter refers to the interaction between grand-mean centered Centered Midpoint by person-centered Pleasant Interaction.
Table 44 Sleep Duration Does Not Moderate the Effects of Hourly Pleasant Interactions on Hourly PA

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Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Centered Duration refers to person-centered sleep midpoint on a given night. Cen Duration*Cen Inter refers to the interaction between grand-mean centered Centered Duration by person-centered Pleasant Interaction.
Table 45 Sleep Duration Does Not Moderate the Effects of Hourly Pleasant Interactions on Hourly NA

Fixed Effects

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Random Effects

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<td>0.01</td>
<td>0.00</td>
<td>4.42</td>
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</tbody>
</table>
Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Duration refers to a participant’s average sleep midpoint across all available monitoring days. Centered Duration refers to person-centered sleep midpoint on a given night. Cen Duration*Cen Inter refers to the interaction between grand-mean centered Centered Duration by person-centered Pleasant Interaction.
Table 46 Sleep Efficiency Does Not Moderate the Effects of Hourly Pleasant Interactions on Hourly PA

<table>
<thead>
<tr>
<th>Fixed Effects</th>
<th>B</th>
<th>95% CI Lower Limit</th>
<th>95% CI Upper Limit</th>
<th>SE</th>
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<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Level 1: Within-Person, Hourly</td>
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</tr>
<tr>
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<td>0.09</td>
<td>0.20</td>
<td>0.03</td>
<td>14423.64</td>
<td>4.97</td>
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</tr>
<tr>
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<td>0.02</td>
<td>14402.57</td>
<td>-1.65</td>
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<tr>
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<td>0.06</td>
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</tr>
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<td>0.01</td>
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<td>351.69</td>
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<td>0.01</td>
<td>360.33</td>
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<th>95% CI Upper Limit</th>
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<td>4.07</td>
<td>0.000</td>
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</tbody>
</table>
Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency*Cen Inter refers to the interaction between grand-mean centered Centered Efficiency by person-centered Pleasant Interaction.
Table 47 Sleep Efficiency Does Not Moderate the Effects of Hourly Pleasant Interactions on Hourly NA

<table>
<thead>
<tr>
<th>Fixed Effects</th>
<th>B</th>
<th>95% CI Lower Limit</th>
<th>95% CI Upper Limit</th>
<th>SE</th>
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<tbody>
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<td><strong>Level 1: Within-Person, Hourly</strong></td>
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<td><strong>Level 2: Within-Person, Daily</strong></td>
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<td>0.00</td>
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<td>0.01</td>
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</table>
Legend: Baseline Depression refers to the Center for Epidemiological Studies-Depression (CESD) scale total score minus the sleep-related item. Workday Status indicates if the participant was working on the day of sleep assessment. Average Efficiency refers to a participant’s average sleep midpoint across all available monitoring days. Centered Efficiency refers to person-centered sleep midpoint on a given night. Cen Efficiency *Cen Inter refers to the interaction between grand-mean centered Centered Efficiency by person-centered Pleasant Interaction.


Biss, R. K., & Hasher, L. (2012). Happy as a lark: Morning-type younger and older adults are higher in positive affect. Emotion, 12(3), 437.


decrements during a week of sleep restricted to 4-5 hours per night. Sleep: Journal of Sleep Research & Sleep Medicine.


Lauderdale, D. S., Knutson, K. L., Yan, L. L., Liu, K., & Rathouz, P. J. (2008). Self-reported and measured sleep duration: how similar are they? Epidemiology, 19(6), 838-845. doi:10.1097/EDE.0b013e318187a7b0


Wagner, U., Fischer, S., & Born, J. (2002). Changes in emotional responses to aversive pictures across periods rich in slow-wave sleep versus rapid eye movement sleep. Psychosomatic medicine, 64(4), 627-634.


